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# PROBABLE CAUSE: A DECISION MAKING FRAMEWORK

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August 1984





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	> Causal inference is an essential cognitive activity that combines judg-			
	ments of causation with judgments-under-uncertainty. Thus, causal inference			
	involves judgments of probable cause. A framework for understanding how			
	judgments of probable cause are made is presented that consists of three			
	major elements: (1) We first discuss the concept of a causal background			

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and show how the causal strength of a variable depends on it being a deviation or difference in a background. This has implications for understanding causes vs. conditions, the differential salience of explanations, and molar vs. molecular explanations; (2) We discuss various cues-tocausality such as covariation, temporal order, contiguity in time and space, and similarity of cause and effect. In doing so, we show how these cues can conflict with probabilistic ideas. A model for combining the background and cues in determining the strength of an explanation is proposed that includes the construction of a causal chain between an effect and its presumed cause. The model is used to discuss a wide range of studies on causal judgments and explicates methodological issues such as spurious correlation, "causalation," and causal inference in case studies; (3) The discounting of an explanation by specific alternatives is discussed as a special case of the sequential updating of beliefs. This leads to a consideration of various factors that have not previously been considered important in the discounting process. Finally, we extend our approach to consider multiple causation; in particular, conjunctive explanations.

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To provide a framework for reviewing the relevant literature on how judgments of probable cause are made, we draw upon several approaches to the study of causality; specifically, work in attribution theory (Heider, 1958; Kelley, 1973), methodology (Cook & Campbell, 1979), and philosophy (Mackie, 1974). We stress that each of these areas has been treated in detail elsewhere and we make no claim to providing exhaustive treatments here. Rather, the focus is on linking causal inference with research on judgments-under-uncertainty.

#### General approach

We assume that judgments of probable cause are based on the judged strength of the causal link between some suspected cause, X, and some effect, Y. Moreover, we argue that the strength of a causal link is a function of three factors: (1) a causal background or context; (2) various cues-tocausality (such as temporal order, covariation, contiguity, and similarity of cause and effect); and, (3) a mechanism for discounting causal strength when specific alternatives are considered. To illustrate, imagine that a watch face has been hit by a hammer and the glass breaks. How likely was the force of the hammer the cause of the breakage? Since no explicit context is given, an implicitly assumed neutral context is invoked in which the cues-tocausality point strongly to a causal relation; i.e., the force of the hammer precedes the breakage in time, there is high covariation between glass breaking (or not) with the force of solid objects, contiguity in time and space is high, and there is similarity in the length and strength of cause and effect. Moreover, it is difficult to discount the causal link since there are few specific alternatives to consider. Now imagine that the same events occurred in a watch factory. In this context, the cause of the breakage is more often judged to be a defect-in-the-glass (Einhorn & Hogarth, 1982a). We

#### Probable Cause: A Decision Making Framework

How do people judge the likelihood that variable X causes outcome Y? The complexity of the answer to this question is attested to by the size and diversity of the literature on causality in a wide variety of disciplines; e.g., philosophy (Bunge, 1979 and references), law (Hart & Honoré, 1959; Fincham & Jaspars, 1980), medicine (Shapiro, 1960; Susser, 1973), statistics (Suppes, 1970; Cohen, 1977; Toda, 1977), psychology (Michotte, 1946; Piaget, 1974). From our perspective, much of the difficulty in assessing causality is due to the fact that judgments of causation are typically judgments of probable cause; i.e., people make causal inferences which take place under uncertainty. Indeed, many practical inferences involve the combining of causal and uncertainty judgments. For example, predictive judgments are typically influenced by one's causal model of how particular variables produce the outcome of interest; diagnostic judgments are concerned with determining the likely cause of outcomes, events, symptoms, and the like; attributions of blame and responsibility rest on judging who (or what) was the likely cause; and so on. Our basic contention is that uncertainty and causality are intertwined in a great many situations and people use strategies in making causal inferences that reflect both factors. Indeed, it is the combination of these two modes of thought that is the focus of this paper. However, as often happens when different systems are combined, conflict and inconsistency can occur. In particular, conflict between causal and probabilistic reasoning can lead to judgments of probable cause that violate probability and statistical theory. We therefore consider such cases because they highlight the various characteristics of each system and the difficulty of integrating one with the other.

net strength of evidence that X causes Y. More formally, denote  $S_k(X,Y)$  as the net strength of X for Y after considering k specific alternatives (k = 0, 1, ..., K). Furthermore, let s(X,Y) be the gross strength of X for Y; i.e., its plausibility or strength before specific alternatives are considered (note that  $S_0(X,Y) = s(X,Y)$ ). The gross strength of X for Y is determined by the causal background and the cues-to-causality, as will be discussed in detail below. Furthermore, denote  $Z_k$  as the kth alternative causal explanation and let  $s(Z_k,Y)$  be the gross strength of  $Z_k$ . The general model is then given by,

$$S_{k}(X,Y) = F[s(X,Y), s(Z_{k},Y)]$$
 (1)

where,  $S_{K}(X,Y)$  is increasing in s(X,Y) and decreasing in  $s(Z_{K},Y)$ . Thus, the net strength of X for Y increases with the gross strength of X, and decreases with the strength or plausibility of competing alternatives.

To illustrate, reconsider the initial watch-hammer scenario and contrast the net strength of the "force of the hammer" explanation with the net strength of any single explanation for the following questions:

1. Why are the outer rings of Saturn braided?

2. Why was Ronald Reagan elected President in 1980?

For the first question, it is difficult to generate a single explanation, thus suggesting its gross strength is low. However, although there are few competing explanations, net strength remains low. For the second question, there are many strong explanations (e.g., the situation of the economy; the rise of the moral majority; the unresolved Iranian hostage problem; etc.). Therefore, while the gross strength of these are high, the net strength of any single one is low precisely because the others are highly plausible alternatives. On the other hand, the watch-hammer question leads to high net strength since the explanation is strong and there are few plausible

consider this in detail below but note that although the cues have not changed, the background shift affects their interpretation as well as the strength of competing explanations.

The interdependence of cues and alternatives on the causal background suggests an analogy between judgments of probable cause and perceptual judgments. In particular: (1) The importance or strength of information in perception depends on the background or field against which it is perceived. For example, object salience involves a figure/ground relation that can be changed by appropriate shifts in the ground as well as in the figure. Similarly, we view the strength of evidence in causal judgment as being highly dependent on an assumed causal background or field; (2) The strength of evidence for a particular causal candidate can be viewed as similar to its "signal strength" relative to a field of competing alternatives. Moreover, as in perception, each potential cause (signal) gives off noisy cues such that a particular pattern can be diagnostic of more than a single category (cf. Campbell, 1966). The importance of this is that the strength of evidence for a particular causal candidate X is seen as its net strength; i.e., how well the evidence supports X as opposed to its competitors; (3) Causal judgments often involve a constructive aspect in which prior theory and expectations are brought to bear on linking suspected causes and effects. In analogous fashion, the importance of expectations and the constructive nature of "achieving" the object are well established in perception (cf. Garner, 1966); (4) Causal inference often occurs with great speed and a corresponding lack of awareness of the underlying process. This is also true of perception.

The factors that comprise probable-cause-judgments can be expressed in a general model that also provides a structure for discussing later developments. This model assumes that judgments of probable cause are based on the

alternatives. In short, it is argued that like good patterns, good explanations have few alternatives (Garner, 1970); or, to be more precise, whereas good explanations imply few strong alternatives, the lack of strong alternatives does not imply good explanations.

While equation (1) is too general to be useful in an operational sense, it shows that an understanding of judgments of probable cause requires:

(a) a model of how the gross strength of an explanation is determined; and

(b) a model of how the gross strength of an explanation is discounted by alternatives.

#### Plan of the paper

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We organize our discussion and review around the various issues raised by equation (1). Specifically, we first discuss the concept of a causal background and show how the causal strength of a variable depends on it being a deviation or difference in a background. Second, we discuss the causal cues of covariation, temporal order, contiguity in time and space, and similarity of cause and effect. In doing so, we show how these cues can conflict with probabilistic ideas. Third, a model for how the background and cues combine in determining the gross strength of an explanation is presented. The model highlights the constraints and conflicts between cues and provides a framework for integrating much research in causal inference. Fourth, a model for discounting the gross strength of an explanation on the basis of alternatives is given. The model serves to illustrate the types of problems that must be addressed in the discounting process. Fifth, our general approach is extended to deal with multiple causation; in particular, conjunctive explanations. Finally, various descriptive and normative implications of our framework are discussed.

#### The causal background

Much research indicates that processes of perception and judgment are sensitive to differences or deviations from present states, adaptation levels, and reference points (e.g., Helson, 1964; Kahneman & Tversky, 1979). Moreover, this sensitivity also applies to the types of events that trigger the asking of causal questions. Specifically, we believe that events of causal interest are those that are unusual, abnormal, or unlikely (cf. Hastie, 1984). Thus, one rarely seeks the cause of why one feels "average," why traffic flowed normally, or why some accident is typical. To be sure, the need for explanations can be aroused vis-à-vis normal events. However, this is most likely to happen when those events violate expectations and are therefore seen as unusual after all. For example, we might want to know why traffic flowed normally if major highway improvements were just completed, or why we feel "average" after hearing about a death in the family. Therefore, causal inference is often invoked to make sense of various kinds of deviations. However, it is important to note that the meaning of a deviation is itself crucially dependent on some assumed background or field. Indeed, even averages can be made unusual with the appropriate shift of background-consider Oscar Wilde's statement that "moderation shouldn't be taken to extremes."

We now consider how the background affects judgments of probable cause and discuss two related issues: (1) the role of the background in distinguishing causes from conditions; (2) how the background makes alternative causes more or less salient.

(1) It has been argued that causal relevance is generally related to the degree that a variable is a difference-in-a-background (Mackie, 1974). By this is meant that factors that are part of some presumed background are

judged to be of little or no causal relevance. For example, does birth cause death? While the former is both necessary and sufficient for the latter (and thus covaries perfectly with it), it seems odd to consider one the cause of the other. The reason is that death presupposes that one has been born. Therefore, birth is part of the background and its causal relevance is low. However, not all factors that are differences-in-a-background are seen as causally relevant. In particular, one must distinguish between causes and conditions. To illustrate the distinction, consider diseases that are generally confined to particular segments of the population (e.g., children, or the elderly). It seems peculiar, for example, to say that Alzheimer's disease is caused by being old. Rather, being old suggests various enabling factors that, when conjoined with some other event(s) (e.g., virus, bacteria, etc.), produces the disease. Indeed, being old may be a necessary condition for the disease, but not its cause. As we argue below, the distinction between particular causes and conditions is highly dependent on some assumed background. However, causes can generally be distinguished from conditions in that: (a) Events are more causal than standing conditions (viruses are more causal than being old); (b) Intrusive events are more causal than events that generally occur; (c) Something abnormal or wrong is more causal than what is normal and right (e.g., the car accident was caused by the person veering to the left, not by the other person who drove straight ahead).

Now consider how a shift in background can change a probable cause into a condition, with a corresponding reduction in its causal strength. Recall our earlier example of a watch-face being hit by a hammer. In the original context, a hammer that hits glass is an intrusive event that does not generally occur while a defect-in-the-glass is a standing state. However, in a watch factory, the hitting of glass by hammers generally occurs while a defect is

something abnormal or wrong. Hence, in this context, the hitting of glass becomes more of a condition or standing state and the explanation loses much of its causal strength. Similarly, the defect explanation gains in causal strength due to its abnormality in these circumstances. However, it is possible to decrease the relative causal strength of the defect explanation by making it less abnormal. For example, consider that all of the watch faces were being broken in the testing procedure. This suggests that the force of the hammer was too great (it being inappropriate for a testing procedure), and its causal strength is thereby increased.

The importance of background shifts on causes vs. conditions is not limited to physical causation. Indeed, consider the work on actor vs. observer differences in attribution theory (Jones & Nisbett, 1972; Watson, 1982 for a recent review). The basic finding is that whereas observers tend to attribute the causes of an actor's behavior to the dispositional traits of the actor, actors tend to attribute the causes of their own behavior to situational variables. Note that from the actor's perspective, his/her own traits are standing states, while situations are more intrusive and hence more causal. From the observer's perspective, situations are seen as standing states and the dispositions of actors are more intrusive. In fact, this asymmetry was noted by Jones and Nisbett (1972) who stated, "For the observer behavior is figural against the ground of the situation. For the actor it is the situational cues that are figural and are seen to elicit behavior" (p. 93). In our analysis, therefore, much should depend on the familiarity of observers with particular actors (e.g., if person A is well known to the observer, A's dispositions may be seen as standing states), as well as the actor's familiarity with particular situations. And indeed, although we know of no studies that have addressed the latter prediction, some evidence exists

supporting the former (Goldberg, 1981, but see Watson, 1982). Moreover, a shift in background does not mean that previous causes lose all their strength or that prior conditions are now seen as strong causal candidates. Clearly, changes in causal strength are one of degree and relative effects can vary considerably. In his review, Watson (1982) notes that dispositions generally receive more weight than situations for both actors and observers, but that the relative weight for dispositions is greater for observers than for actors.

The influence of background shifts also clarifies a perennial issue in scientific inquiry--reductionism in causal explanations (causes at a molar level are different from those at a molecular level). If one thinks of the background as analogous to the field of vision under a microscope, then shifts in magnification of the lens define different fields. Moreover, since causal relevance is at least a difference-in-the-field, it is obvious that a cause at one level will not necessarily be relevant at another. This microscope analogy makes clear that the "appropriate" level of magnification depends on one's purposes and the extent of one's knowledge of the phenomenon in question. Thus, a biochemist may see the causal link between smoking and lung cancer as due to chemical effects of tar, nicotine, and the like, on cell structure, while an immunologist might see the causal link as due to the suppression of the immune system in controlling diseases in general. However, it should be noted that the level of the field is not totally arbitrary in everyday inferences. Indeed, there is remarkable consensus among individuals as to the appropriate level of the assumed background. On the other hand, where large discrepancies exist in knowledge about a particular topic, as in comparing experts to non-experts, such consensus is often lacking.

(2) In addition to defining differences/deviations and distinguishing between causes and conditions, the causal background plays an important role

in affecting the number and salience of alternative explanations. That is, if one considers the causal background as containing the set of alternatives against which a particular causal candidate is evaluated, then shifts in the background can highlight, or de-emphasize, classes alternatives. For example, imagine the following scenario: Joe is a chemical worker who contracts lung cancer and sues the company for causing his disease. His lawyer argues that the cancer rate of workers in this factory is nine times the national average for workers in comparable industries. Note that the background in this argument is industries of a certain type and the causal argument rests on there being a difference (higher cancer rates) in this background. However, the lawyers for the chemical company may shift the background by arguing that Joe has smoked cigarettes for years, comes from a family with a history of cancer, and so on. Note that the background is now changed to people with certain personal habits and characteristics thereby making these habits and characteristics highly salient alternatives. Moreover, the background shift also changes Y from an unusual event that requires a special causal explanation (working in a chemical plant) to a less unusual event that requires no such explanation.

In addition to shifts in the background, alternatives can be ruled in or out depending on the breadth of the background. In the above scenario, for example, note how Joe's case would be strengthened if it could be shown that the cancer rate in his factory was nine times the rate of other chemical factories making exactly the same product. The reason is that by narrowing the field to chemical plants making the same product, the number of alternatives explanations is reduced, thereby making the difference in the narrowed field more causally relevant. A similar idea has been advanced by Bar-Hillel (1980). In considering the research showing that people ignore base rates in

making probability judgments, she demonstrates that base rates will be used if they are made more specific or if they can be given a causal interpretation. She suggests that both specificity and a causal interpretation increase the "relevance" of information, and thus its use. From our perspective, Bar-Hillel's treatment of relevance is consistent with our view of probable cause; both are increased by specific causal evidence and a narrowing of the background (specificity) which reduces alternatives.

#### Cues-to-causality

Once a particular background or context has been evoked, the strength of a causal link between X and Y depends on various cues-to-causality. We note that the term "cues" has a specific meaning that corresponds with its use in Brunswik's psychology (1952; also see Hammond, 1955; Campbell, 1966). Therefore: (1) The relation between each cue and causality is probabilistic. That is, each cue is only a fallible sign of a causal relation; (2) People learn to use multiple cues in making inferences in order to mitigate against the potential errors arising from the use of single cues; (3) The use of multiple cues is often facilitated by the intercorrelation (redundancy) between them in the environment. This both reduces the negative effects of omitting cues, and aids in directing attention to the presence of others; (4) Although multiple cues can reduce uncertainty in causal inference, conflicting cues can increase it. As we shall see, conflicting cues often require that distinctions be made with respect to the type of causal relation present (e.g., precipitating vs. underlying cause). We consider these issues later, in connection with a model for combining the cues.

In discussing the cues, we consider each at some molar level although we are fully aware that they can be decomposed into more molecular components.

For example, contiguity can be decomposed into temporal and spatial cues and

temporal contiguity can be further divided into the time interval between cause and effect and the regularity of the interval. The importance of considering the elements of each cue will become apparent as we proceed.

The cue of <u>covariation</u> is considered first since it has received considerable attention by numerous authors (Kelley, 1973; Nisbett & Ross, 1980; Crocker, 1981; Alloy & Tabachnik, 1984). We begin by noting that when people judge that X caused Y (or X will cause Y), they rarely mean that X is either necessary or sufficient for Y. For example, if a fire inspector said that a short-circuit (X) caused a house fire (Y), it is clear that X is neither necessary nor sufficient for Y. Rather, it means that X, when conjoined with a particular set of conditions, leads to Y (e.g., flammable material near the short circuit, no sprinkler system, etc.). If we denote W as representing flammable material near the short-circuit and Z as the absence of a sprinkler system, then define  $\alpha$  as the scenario in which X and W and Z occurred; i.e.,  $\alpha = (X \cap W \cap Z \mid B)$  where, B = causal background. After Mackie (1965), we call  $\alpha$  a minimally sufficient condition for Y if,

$$p(Y|\alpha) = 1 \quad \text{but}, \quad p(\alpha|Y) \neq 0,1 \tag{2}$$

Thus, given  $\alpha$ , Y always follows; however, given Y,  $\alpha$  is neither certain nor impossible. The scenario  $\alpha$  is said to be minimally sufficient if the deletion of any of its conjuncts makes  $\alpha$  no longer sufficient. Furthermore, it is important to note that the sufficiency of  $\alpha$  always presupposes some causal background, otherwise the number of conjuncts in  $\alpha$  would be very large. For example, it is assumed that oxygen is part of the causal field since there can be no fires without its presence. Indeed, since oxygen is a standing state, it can be seen as a necessary condition and thus part of the background in this context.

If we now consider the short-circuit as the probable cause of Y, note

that X is necessary for the scenario a but it is not sufficient; i.e.,

$$p(X|\alpha) = 1$$
 but,  $p(\alpha|X) \neq 0,1$  (3)

We can now say that X is an insufficient but necessary part of a complex scenario (a), which is itself unnecessary but sufficient for Y (INUS for short). Mackie notes that the statement, "the short-circuit caused the house fire," implies that: (a) X is at least an INUS for Y; (b) X occurred; (c) the other conjuncts occurred; (d) all minimally sufficient conditions for Y not having X in them were absent on the occasion in question.

The above conceptualization provides a useful and important link between probable causes and covariation. Consider a 2  $\times$  2 table in which X and  $\overline{X}$  are

## Insert Table 1 about here

crossed with Y and  $\bar{Y}$ . The upper left-hand cell (cell A) contains the number (and frequency) of scenarios  $(\alpha_{\bar{Y}\cap X})$  in which X conjoins with various conditions to cause Y. The upper right hand cell (cell B) contains scenarios in which X occurs but the remaining conditions are insufficient for Y  $(\alpha_{\bar{Y}\cap X})$ . This can result if there are too few enabling conditions to allow Y's occurrence; or, conditions exist that cancel or prevent X from causing Y. For example, imagine that X was a particular virus, Y was some disease, and W was an antibody that existed in some proportion of the population. If W counteracts X, then the conjunction  $(X\cap W)$  does not produce the disease. We denote the frequency of  $\alpha_{\bar{Y}\cap X}$  scenarios as indicating the "conditionality of causation." That is, the larger this cell, the more X is either sensitive to cancellation or the more it has to conjoin with other conditions to produce Y.

Now consider the lower left-hand cell (cell C), which represents the scenarios  $\alpha_{Y \cap X}$ . This cell contains the minimally sufficient conditions for Y in which X is absent and thus indicates the existence of alternative causes

TABLE 1
Contingency Table Representing Causal Relations

	E	Effect	
	Y	Ÿ	
3	α <sub>Y</sub> ηχ	$lpha_{\overline{Y}\cap X}$ conditionality	
Suspected	(A)	(B)	
Cause	α <sub>γΩχ</sub> multiplicity	$^{lpha}ar{\mathtt{Y}}\capar{\mathtt{X}}$	
	(c)	(D)	

of Y. To contrast this source of uncertainty with the "conditionality of causation," we consider the frequency of  $\alpha_{\begin{subarray}{c} Y \cap X\end{subarray}}$  scenarios as representing the "multiplicity of causation." Finally, the lower right-hand cell contains those scenarios in which neither X nor Y occurs,  $\alpha_{\overline{Y}\cap \overline{X}}$  . This cell is problematic since it implicitly defines the context in which X and Y are being considered. For example, consider the relation between smoking (X) and lung cancer (Y). Cell A contains the minimally sufficient scenarios for Y in which X occurs; Cell B contains insufficient scenarios for Y in which X occurs; Cell C contains sufficient scenarios for Y that do not contain X; Cell D contains the scenarios in which no smoking conjoins with other conditions to lead to no lung cancer. In this latter cell, questions arise as to what objects to include as having  $\ddot{x}$  and  $\ddot{y}$ . For example, should we allow children to be included (after all, they don't smoke and don't have lung cancer)?; if not, at what age are we to define the relevant population?; Should the analysis be different for men vs. women; people with a history of cancer in the family vs. those without, etc.? What is important to note is that covariation, by its very nature, implies a reference population and thus some background against which the relation should be assessed.

The above conceptualization has several general implications. First, the traditional notions of necessity and sufficiency of causation can be represented by covariation in that a necessary cause is one that is in all minimally sufficient scenarios for Y (implying that the C cell is empty); a sufficient cause is one that always conjoins with conditions to produce Y (implying that the B cell is empty). Second, the idea of considering scenarios of events as comprising the "raw data" for judging covariation is consistent with the use of this cue in singular cases; i.e., where the data consists of a single instance. For example, imagine that you feel ill today

and recall that you ate fish last night. Since there is only one observation, covariation might be thought irrelevant. However, one can imagine (or remember) scenarios involving illness without eating fish; and, one can imagine (or remember) scenarios in which a fish meal produces (or produced) no ill effects. Furthermore, even if one's imagination or experience is limited, one is aware of the uncertainties associated with both the multiplicity and conditionality of causation. That is, one is aware of one's incomplete knowledge regarding: (a) all the conditions that can conjoin with X to produce Y; and, (b) the alternatives that can cause Y in the absence of X. Put succinctly, people know that they don't have complete causal knowledge. Therefore, the awareness of incomplete knowledge provides the basis for why causal judgments are generally judgments of probable cause.

We now consider some specific implications of a scenario-based approach to causation for discussing the literature on judgments of covariation. To begin, we conceive of covariation judgments (denoted as  $Q_1$ ) as resulting from a weighted linear combination of the cell frequencies from a 2 × 2 table. Thus,

$$Q_1 = \sum_{i=1}^4 \beta_i q_i \tag{4}$$

where,  $q_1 = \alpha_{\widehat{Y} \cap X}$ ;  $q_2 = \alpha_{\widehat{Y} \cap X}$ ;  $q_3 = \alpha_{\widehat{Y} \cap \widehat{X}}$ ;  $q_4 = \alpha_{\widehat{Y} \cap \widehat{X}}$ ;  $\beta_1 = \text{weight given to}$   $q_1$ .

Equation (4) provides a simple and convenient way of summarizing much of the research on covariation judgments. For example, Smedslund (1963) and Jenkins and Ward (1965) showed that their subjects' judgments were based almost exclusively on  $\alpha_{X \cap X}$  (i.e.,  $\beta_1 > 0$ ,  $\beta_2 = \beta_3 = \beta_4 = 0$ ); Ward and Jenkins (1965), however, changed the way information was presented to subjects (from sequential to intact displays), and found different patterns of use

(many subjects ignored disconfirming evidence, i.e.,  $\beta_2 = \beta_2 = 0$ ; many other subjects weighted all cells); Binhorn and Hogarth (1978) noted that information is frequently absent from real-world tasks such that  $\beta_1$ ,  $\beta_2 > 0$  but  $\beta_{\alpha} = \beta_{A} = 0$ . Since much of the research on covariation judgments compares intuitive responses with some statistical measure of covariation (usually the correlation coefficient -- in which the four cells are equally weighted), the ignoring of certain cells is often taken as evidence that people have a poor intuitive concept of correlation. However, numerous studies have also shown that people can and do make use of all of the q,'s that are available (Alloy & Abramson, 1979; Crocker, 1981). A recent meta-analysis speaks directly to this issue. Lipe (1982) has shown that when subjects' responses were regressed onto the four data cells over a number of studies, the weights for all four  $q_4$ 's were significant and in the expected directions  $(\beta_4, \beta_A > 0;$  $\beta_2,\beta_3$  < 0). Therefore, the question is not whether people sometimes ignore relevant information but rather, under what conditions is particular information ignored or not? For example, Crocker (1982) has demonstrated that the type of question asked can greatly affect attention, and thus the amount of weight given to each cell (also see, Arkes & Harkness, 1983).

We believe that when people are asked to judge the covariation between two variables, they may interpret the question in a causal way (the tendency to equate correlation with causation is very strong; otherwise, why warn students who take statistics courses that correlation does not imply causation?). Moreover, if questions are thought to reflect causal relations, one would expect greater use of all four cells since the uncertainties (unknown multiplicity and conditionality) of a causal relation are more salient than in covariation per se. In fact, Schustack and Sternberg (1981) showed that when subjects were given information in the form of the  $q_1$ 's and

were asked to give causal judgments, a regression model like equation (4) yielded results showing that  $\beta_1$ ,  $\beta_4$  > 0 and  $\beta_2$ ,  $\beta_3$  < 0. In another study (Waller & Pelix, 1982), subjects were asked to judge the same information by answering both a causal and a correlational question. In accord with our view that covariation is a fallible cue to causality, they found a moderate but significant correlation between the two types of judgments (r = .57).

If causal rather than covariation questions direct attention to more  $\mathbf{q}_{\mathbf{i}}$ 's, one implication is that the judged causal strength of two variables should be closer to statistical measures of correlation than are judgments of covariation. However, we know of no research that speaks to this issue. On the other hand, causal judgments based on covariation data are prey to attentional shifts in their own right. Specifically, causal questions can be asked in a future or forward direction (how likely will X cause Y?), a past or backward direction (how likely was Y caused by X?), or a temporally neutral manner (how likely are X and Y causally related?). In forward-causalinference, the focus of attention is on the conditionality of causation (cells A and B) since the uncertainties about the conditions that enable X to produce Y are made salient. Indeed, Schustack and Sternberg (1981) asked subjects to make forward-causal-inferences and found that the regression weight for cell B was higher than for cells C and D (cell A had the highest weight; all weights were significantly different from zero). In backward-causal-inference, the focus of uncertainty is on the unknown multiplicity of causation (cell C). In fact, some philosophers have implicitly assumed a backward inference direction in discussing causation. Thus, Mackie (1974) notes that in assessing whether X caused Y, one implicitly asks the counter-factual question, "Would Y have occurred if X had not?" Note how this focuses attention on the multiplicity of causation by asking one to imagine minimally sufficient conditions for Y

that do not contain X (i.e.,  $\alpha_{V \cap X}$ ).

The idea that people can differentially weight the  $q_1$ 's due to attentional shifts is an important yet virtually unresearched area in judging probable cause. In particular, the notion that the direction of inference (forward/backward/neutral) can affect the relative sizes of the  $\beta_1$ 's seems especially promising. Such research, for example, should be able to resolve the issue of whether "necessity" or "sufficiency" of causation is more strongly related to judged causal strength. According to our conceptual scheme, necessity should be most important in backward inference (would Y have occurred if X had not?), but sufficiency should be most important in forward inference (could  $\overline{Y}$  occur if X does?). When causal questions are asked in a temporally neutral way, both necessity and sufficiency are likely to play a role (i.e., cells B and C should be more equally weighted).

In addition to the direction of inference, another factor is likely to affect causal judgments via the covariation cue. We indicated above that cell D ( $\alpha_{\overline{Y} \cap \overline{X}}$ ) is often problematic in that the definition of what constitutes the absence of X and Y depends on some assumed background or reference population. Consistent with this is the fact that cell D is rarely weighted as heavily as the other three cells. However, there is a sense in which this cell is important in that it raises the issue of the <u>robustness</u> of the X,Y relation over other backgrounds or populations. For example, imagine a high correlation between a particular diet and heart disease in the U.S., but a lack of any such correlation in Western Europe. We would expect that in judging the causal strength of X and Y, robustness will be seen as an important cue-to-causality. Indeed, an interesting and unresearched question concerns whether, or to what degree, people are willing to trade-off high covariation (in one setting) with lower covariation that is robust over several backgrounds.

Temporal order, contiguity, and similarity

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We now consider the cues of temporal order, contiguity in time and space, and similarity of cause and effect. Temporal order (denoted Q<sub>2</sub>) is discussed first. The importance of temporal order in causal judgments seems obvious since it is essential for determining which of two variables in a relation is cause and which is effect. However, whereas temporal order greatly affects causal judgments, this cue has no role in formal probability theory. This suggests that there is likely to be much conflict and confusion in probability judgments when temporal order is salient. We illustrate this conflict by considering three issues: the confusion of joint and conditional probabilities, causal vs. diagnostic probabilities, and the confusion of inverse probabilities (Dawes, undated).

An important feature of temporal order is the speed and facility with which it is used—often without explicit awareness. This is particularly the case in the interpretation of language; consider the conjunction "and," which frequently implies temporal order in everyday English (Strawson, 1952). However, the word "and" is used in formal probability to denote a conjunction of events, irrespective of temporal order. Therefore, how should one understand a question like, "What is the probability of going into the supermarket and buying some coffee?" If we let S = going into the supermarket, and K = buying coffee, the probability of S and K could be interpreted in several ways. Whereas a statistician would represent the question as  $p(S\cap K)$  and ignore the temporal meaning of "and," others may perceive the question as requiring the conditional probability, p(K|S). That is, one could consider the sequence of events as being important and condition the uncertainty of the later event p(K) on the earlier one p(K). Indeed, to direct attention to the conjunction of the events, it might be helpful to reverse S and K in order

to break the implied time order, i.e., "What is the probability of buying some coffee (K) and going into the supermarket (S)?" An experiment to test this assertion was performed using graduate students with at least one statistics course. One group  $(n_1 = 24)$  was asked to choose how they would represent "S and K" probabilistically, while a second group  $(n_2 = 24)$  was asked to represent "K and S". Subjects chose from either  $p(S\cap K)$ , p(K|S), p(S|K), or "none of the above." The results showed an increase for  $p(S\cap K)$  when the time order was reversed (58% to 75%). Of further interest was the finding that 38% of the subjects chose p(K|S) in the first group (in accord with the natural order of the events) while no subjects chose p(K|S) in the second group. Clearly, temporal order is an important cue that is difficult to ignore, even when it may be appropriate to do so.

A further example of the importance of temporal order in probability judgments has been discussed by Tversky and Kahneman (1980). They argue that people judge the probability of data that has causal significance as being greater than data that have diagnostic value, even when the information is equally informative from a statistical point of view. For example, compare the probabilities that: a girl has blue eyes (Y) if her mother has blue eyes (X) vs. a mother has blue eyes if her daughter has blue eyes. Although p(Y|X) = p(X|Y), making X and Y equally informative, many subjects judge the causal/forward probability as greater than the diagnostic/backward one (i.e., p(Y|X) > p(X|Y)). Tversky and Kahneman also demonstrate that when temporal cues are ambiguous, the causal interpretation of data tends to receive disproportionate weight such that elementary rules of probability can be violated.

The above suggests that temporal order has a straightforward effect on probability judgments. However, the situation is more complex; in particular,

people often have difficulty in distinguishing between p(Y|X) and p(X|Y)when X and Y are not equally informative. This "confusion of inverse probabilities" (Dawes, undated) seems to contradict the importance of temporal order as a cue that unequivocally leads to causal probabilities being judged larger than diagnostic ones. However, we now discuss when the confusion is most likely to occur and argue that it is due to the ambiguity of temporal cues in the task. To illustrate, consider the case of mammography and breast cancer (Eddy, 1982). For women with breast cancer (C), the probability of a positive mammogram (M) is .79 (p(M C) = .79). Eddy reports that most physicians misinterpret the statements about the test and estimate the probability of breast cancer given a positive test to be approximately .75 (p(C|M) = .75). "When asked about this, the erring physicians usually report that they assumed that the probability of cancer given that the patient has a positive X-ray . . . was approximately equal to the probability of a positive X-ray in a patient with cancer . . . . The latter probability is the one measured in clinical research programs and is very familiar, but it is the former that is needed for clinical decision making. It seems that many if not most physicians confuse the two." (Eddy, 1982, p. 254.)

Under what conditions is the confusion most likely to occur? From our perspective, first note that cancer causes the positive test result and not vice-versa. Therefore, denote cancer as the cause (X) that temporally precedes the test as an effect (Y). However, the test result is known first in time and it is used to predict the cause, X. The peculiarity here is that the test predicts a prior state (cancer) that presumably existed before the test and which caused the test result itself. Hence, this situation has a loop-like structure that makes the temporal order of events particularly confusing. This loop-like structure is shown in Figure 1.

### Insert Figure 1 about here

Note that the causal probability, p(Y|X) is not predictive in the usual sense; i.e., while having cancer predicts the test, this hardly seems relevant to the practical matter. On the other hand, the test (which is known first in time), is used to predict the disease that preceded it (since the disease causes the test result). Therefore, there are two temporal cues in problems of this type: causes precede effects, but, effects are seen first and used to predict their prior causes. Under such conditions, confusion of inverse probabilities is quite understandable. To make matters worse, in statistical jargon, the "diagnosticity" of a datum (D) to some hypothesis (H) refers to p(D|H). However, in ordinary discourse, the diagnostic value of D for H means how well does D predict H (i.e., p(H|D)). Therefore, to avoid such confusion, it would be useful when discussing p(D|H) to say that D is symptomatic rather than diagnostic of H.

The next cue to be considered is <u>contiquity</u> (Q<sub>3</sub>). The extent to which events are contiguous in time and space is an important cue to causality as evidenced in the work of Michotte (1946). Siegler has also shown that for young children (5-6 years old), temporal contiguity is a very strong cue for inferring causality (Siegler & Liebert, 1974; Siegler, 1976). Moreover, these studies show that older children are less dependent on contiguity alone, being able to make use of multiple cues. Nevertheless, contiguity remains an important cue for directing attention to contingencies between variables, and such contingencies may then be considered as to their causal significance. When temporal and/or spatial contiguity is low (or, temporal contiguity is erratic), inferring causality becomes more difficult. That is, in the absence of contiguity, relations are hard to develop unless one uses intermediate causal models to link the events (see the next section). For instance, the

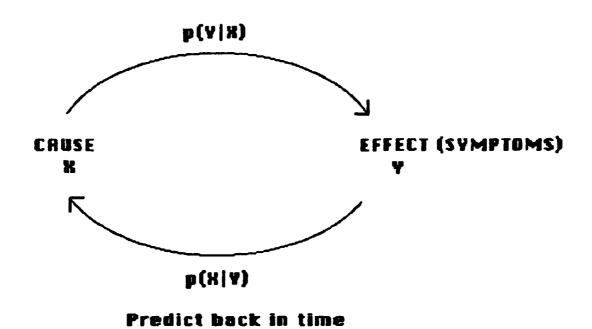


Figure 1. Loop-like structure for confusion of inverse probabilities

temporal gap between intercourse and birth requires some knowledge of human biology and chemistry to maintain the links between those events.

While the above seems obvious, it should first be noted that the sign and form of the relation between contiguity and judgments of probable cause depends on one's knowledge of a particular situation. For example, imagine that one has advertised a product and sales go up dramatically the next day. If one believes that advertising works by a gradual diffusion process, the sales increase may not be attributed to the ad precisely because the events occurred too close in time. On the other hand, contiguity could be seen as monotonic with causal strength by others with different theories, or by the same person in another context. Second, contiguity is particularly prone to conflict with other cues, which can then require compromises of several sorts. While we consider this in more detail in the next section, note how the belief that increased contiguity in time leads to greater causal strength, can conflict with temporal order if cause and effect occur simultaneously. If temporal order has priority, the relation between contiguity and judgments of probable cause is not monotonic.

The cue of similarity (denoted  $Q_4$ ) is important to causal judgments. Like covariation, similarity can be modeled as a function of its elements, some of which add, and some subtract, from its strength. That is, following Twersky (1977), similarity judgments can be defined as a weighted linear function of the common elements of two objects (cf. cell A in covariation) minus the distinctive elements of each (cf. cells B and C). However, to extend this conception of similarity from objects to causes and effects, it is necessary to specify the common and distinctive elements of the latter. These can be considered at several levels. First, there is a long-standing notion that cause and effect should exhibit some degree of physical resemblance.

Mill noted that this is a deeply rooted belief that, "not only reigned supreme in the ancient world, but still possesses almost undisputed dominion over many of the most cultivated minds" (cited in Nisbett & Ross, 1980, p. 115). Mill thought that such a belief was erroneous and many cases exist in which physical resemblance has been misleading. For example, Nisbett and Ross (1980) point out that physical resemblance was the cornerstone of a medical theory called the "doctrine of signatures" whereby cures for diseases were thought to be marked by their resemblance to the symptoms of the disease. Thus, the curing of jaundice was attributed to a substance that had a brillant yellow color (see also Shapiro, 1960; Shweder, 1977). However, whereas physical resemblance may be a cue of low validity, it does not mean it has no validity. Indeed, there are many examples of where it is useful.

At a second level, one can consider similarity based on the <u>congruity</u> of the length and strength of cause and effect. That is, if the effect of interest is large (i.e., is of substantial duration and/or magnitude), people will expect the cause(s) to be of comparable size. For example, the germ theory of disease advanced by Pasteur must have seemed incredible to his contemporaries in that people were asked to believe that invisible creatures caused death, plagues, and so on. In the same way, it is equally difficult for many to believe that billions of dollars spent on social programs in the '60s and '70s could have had little or no effect, or that long term and complex effects like poverty can have short term and simple causes.

#### Determinants of Gross Strength

Causal chains. While the causal background and cues-to-causality are important in determining the gross strength of an explanation, are other factors involved? Several authors have argued that attributions of physical causation depend on the perception of a "generative" force that links causes

to effects (e.g., Shultz, 1982). This implies a strong mechanistic conception of causation in which effects result from a physical transmission of causal "energy" from X to Y. The importance of this idea is that it implies that events need to be linked via a causal chain so that the force can be transmitted from one link to the next. Conversely, if no connecting links can be made between X and Y, the strength of a causal explanation will be zero. To illustrate, how likely is it that sun-spots (X) cause price-changes on the stock market (Y)? In order to link these events, one needs a causal chain that bridges the spatial and temporal gap (assume that temporal order and covariation both point to a causal relation and that prices are affected six months after sun-spot activity). If someone cannot construct a causal chain to link X and Y, we argue that this is sufficient for zero gross strength. Put differently, the existence of a causal chain is necessary for non-zero gross strength regardless of the levels of the cues. Therefore, causal relations must be "achieved" in the sense that prior knowledge and imagination are needed to construct a schema, scenario, or chain, to link cause and effect. On the other hand, a scenario that does link X and Y may be so weak as to result in a zero or near zero gross strength. For example, imagine the following causal chain: sun-spots affect weather conditions, which affect agricultural production, which affect economic conditions, which affect profits, which affect stock prices. However, before explicating the implausible nature of this chain, we need to consider the role of the cues and background in the construction of such chains.

If a causal chain is necessary for linking cause and effect, what rules govern its construction? Of particular importance is the fact that the cuesto-causality constrain the possible causal chains that can be constructed. This is most obvious in considering the cue of temporal order. That is, if X

does not temporally precede Y, it cannot be a cause of Y. However, the cues of contiguity in time and space, congruity (i.e., similarity of length and strength of cause and effect) and covariation also constrain the linkage process. First, consider contiguity in time and space and recall the sunspots and stock prices example. In order to link these events at all, it was necessary to bridge the spatial and temporal gap by positing a change in weather, etc. However, imagine that price changes occur immediately after sun-spot activity (rather than six months later). Note that the high contiguity in time precludes the weather-economic conditions-scenario since that scenario requires a time delay between X and Y. Hence, in order to link X and Y, another scenario is necessary that is consistent with high contiguity in time (and low contiguity in space). Similarly, imagine taking up smoking and getting lung cancer the next week. It seems highly unlikely that smoking is the cause since a causal chain with high contiguity in time is not easy to construct. Now consider incongruity of cause and effect; i.e., small causes/big effects or, big causes/small effects. To account for incongruity, the linkage process must involve some type of "amplification" in the first instance, and "dampening" in the second. For example, re-consider the incongruity between germs and illness/death, as exemplified by the germ theory of disease. Without knowledge of how germs enter the body, multiply, disseminate throughout the system, and so on, it would be difficult (and was difficult at the time), to accept such a theory.

A summary of the effects of the cues on constraining the type of causal chain necessary to link X and Y is given in Table 2, where the cues of contiguity, and congruity are shown at each of two levels. Constrast, for

### Insert Table 2 about here

example, cells 1 and 4. In the former, the two cues have high values such

TABLE 2

Effects of Contiguity and Congruity on
Constraining the Causal Chain

	Cong	Congruity	
	High	Low	
High	Few links needed (if any).	Links needed to amplify or dampen process. (2)	
Low	Links need- ed to bridge congruity gap.	Links needed to bridge contig- uity gap and amplify or damp- en process.	

that one would expect a model requiring few links, if any. Recall, for instance, the example of a hammer hitting a watch face and causing the glass to break. In the latter (cell 4), both cues are low and therefore place quite strong demands on the type of causal chain that can meet specifications.

There is an important connection between the construction of a chain that meets the constraints of the cues and the strength of that chain. To explicate the nature of this relation, we first denote  $Q_L$  as the strength of the causal chain that links X with Y. If one cannot construct a linkage between X and Y,  $Q_L = 0$ . If a causal chain can be constructed, then  $Q_L$  measures the strength of that chain. In order to discuss the factors that determine  $Q_L$ , recall the sun-spot-stock-price causal chain given above:

Sun-spots + weather + agriculture + economy + profits + price-changes X + x<sub>1</sub> + x<sub>2</sub> + x<sub>3</sub> + x<sub>4</sub> + Y

Note that while this scenario meets the constraints in Table 2, it requires a long chain of events to bridge the temporal and spatial gap (we would argue that congruity is not a problem here). Let ½ be defined as the number of links in the constructed chain, where ½ is equal to the number of intermediate variables in the chain (small x's) plus one. Thus, in the above chain there are five links. What determines the strength of each link, and, how do the individual link-strengths combine to determine the strength of the whole

Before individual link-strength can be determined, the constraints of temporal order, contiguity, and congruity must have been met. Therefore, we argue that it is the covariation between variables at each link that determines link-strength. For example, the link between sun-spots and weather is due to the covariation of X with  $x_1$ ; the weather-agricultural production link is due to the covariation of  $x_1$  with  $x_2$ ; and so on. Now consider how

chain (i.e., Q.)?

the individual links combine to determine the strength of the causal chain,  $Q_L$ . To examine this, let  $c_j$  be the covariation of variables at the jth link in the chain (j=1, 2, ..., l) and assume that the relation between the  $c_j$ 's and  $Q_L$  is as follows:

$$Q_{L} = \prod_{j=1}^{\ell} c_{j} \tag{5}$$

By assuming a multiplicative function, equation (5) implies the following: (1) The strength of a chain is at best, equal to its weakest link. Thus, if  $c_1$  has the lowest link-strength in the chain,  $Q_L$  is at best equal to  $c_1$  (if all other links are 1). Indeed, note that if any  $c_j = 0$ ,  $Q_L = 0$  regardless of the strength of the other links. Also note that since  $c_j$  is between 0 and 1, the longer the chain (£), the lower the strength of the whole chain (since one is multiplying fractions). Therefore, equation (5) accords well with some basic intuitions regarding the relations between the number and strength of links in determining the strength of the chain; (2) The effects of covariation at the link-level and the number of links in a chain can be more easily understood by approximating equation (5) using the average covariation between links,  $\overline{c}$ , for  $c_i$ . Thus,

$$Q_{L} = \prod_{j=1}^{\ell} \overline{C} = \overline{C}^{\ell}$$
 (6)

where,

$$\overline{c} = (1/2) \sum_{j=1}^{2} c_{j}$$

Equation (6) makes clear why the sun-spot-stock-price scenario is implausible on the basis of  $Q_L$ . Since  $\ell=5$  in that scenario, even if  $\overline{c}=.7$ ,  $Q_L$  is only .17. Therefore, long chains are generally weaker than short ones. However, equation (6) also implies that if  $\overline{c}$  is high, long chains can be stronger than short chains with lower  $\overline{c}$ . Indeed, when  $\overline{c}=1$ , the

length of the chain is irrelevant. For example, consider the following scenario:

Mrs. Jones, a middle-aged lady, was looking for her seat at a foot-ball game. While trying to ease past some other spectators on a steep staircase, she lost her balance and fell over. She hit another spectator, who was also off balance. This person, in turn, fell on someone else who unwittingly pushed Mr. Smith, a 70-year old man, against some iron railings. Mr. Smith broke a leg in the incident.

How likely did Mrs. Jones' fall cause Mr. Smith to break his leq? We designate such scenarios as "domino-chains," for obvious reasons. The basic characteristics of these chains is a high  $\overline{c}$ , high  $\ell$ , and high  $Q_{\overline{l}}$ . On the other hand, when  $\overline{c}$  is low, even moderate  $\ell$  leads to a low value of  $Q_L$ ; (3) Equations (5) and (6) suggest that the level of the causal background can also affect  $Q_L$  by changing the number of links in the causal chain. To see this, recall the child's game of asking "why?" after each answer. This game highlights the fact that the links in a causal chain are subject to further analysis at a more reductionistic level. As pointed out earlier, the causal background implies a particular level of explanation and as such, it constrains the type and length of the causal chain used to connect X with Y. In particular, as each link is decomposed into further components, the number of total links ( $\ell$ ) increases. However, unless the  $c_4$ 's also increase (which is possible since the phenomenon may be better understood at lower levels), Q could be seen to decrease. Clearly, the rates at which £ and  $c_{\dot{1}}$  increase (if they do) determine whether  $Q_{\dot{L}}$  is higher or lower at different levels of the causal background.

A combining rule for gross strength. As should be clear from the preceding sections, judgments of gross strength are composed of numerous

factors. To recapitulate, Table 3 lists these factors including some of the assumed relations between them. Note that we have also specified the causal background by a variable  $Q_{\rm R}$  for reasons to be explicated below.

### Insert Table 3 about here

Evidence concerning the manner in which the variables listed in Table 3 combine in affecting gross strength can be found in many areas of psychology. Moreover, this evidence can be conceptualized as illuminating two key issues:

(a) What are the necessary conditions for perceiving causal relations? and (b) How are conflicts between the cues resolved?

(a) Three variables can be considered necessary;  $Q_{\rm R}$ ,  $Q_{\rm C}$ , and  $Q_{\rm C}$ . As discussed previously, if a variable is not perceived as a difference-in-afield, it will not be deemed causally relevant. Similarly, temporal order is a necessary condition since causes must precede effects. Now consider the evidence regarding the necessity of  $Q_{T,\bullet}$ . Several researchers have demonstrated that despite the presence of strong cues-to-causality, people will still fail to make causal attributions if they are unable to perceive a causal link (i.e.,  $Q_L = 0$ ). For example, whereas Michotte (1946) showed that subjects perceived causal relations when the movement of objects after contact was congruent with prior trajectories and/or positions, this was not the case when one object touched the other and the latter changed color, got larger, or rose. Consider further the literature demonstrating the limits of classical conditioning. For instance, although Watson and his colleagues were able to condition little Albert to fear rabbits by pairing the appearance of a rabbit with that of a large noise, they could not produce the same effect when the rabbit was replaced by a block of wood or a cloth curtain (Nisbett & Ross, 1980, p. 104). Similarly, Garcia and his colleagues (Garcia et al., 1968; Garcia et al., 1972; Garcia, 1981) have demonstrated that rats can learn to

# Variables and Assumed Relations in Judgments of Gross Strength

 $Q_{\rm B}$  = degree to which X is a difference-in-the background (0 <  $Q_{\rm B}$  < 1)

 $Q_1 = \text{covariation of X with Y} \qquad (0 \le Q_1 \le 1)$ 

 $Q_2 = \text{temporal order of } X \text{ and } Y$  (0, 1)

 $Q_3$  = contiguity in time and space

 $Q_4$  = similarity of cause and effect congruity physical similarity ( $Q_4$ )

 $Q_{L}$  = causal chain strength (0  $\leq$   $Q_{L} <$  1)

=  $\begin{cases} 0 & \text{if no construction} \\ \frac{1}{3} & \text{otherwise;} \approx \overline{c}^{2} \end{cases}$ 

where, l = number of links in the causal chain
= f(Q<sub>3</sub>, congruity)

c<sub>j</sub> = covariation at jth link

l
C = \( \) c /l

associate, after one trial, distinctive tasting food and a gastro-intestinal illness (induced by X-rays) several hours later, i.e., despite a large temporal gap. However, the rats do not learn to associate a differential shape of food to the illness, when the taste is familiar. In a provocative review of this and other literature on learning, Seligman (1970) suggested that organisms are differentially prepared to learn different types of relations. Specifically, Seligman argued that stimuli can be arranged along a continuum of preparedness, from "contra-prepared" through "prepared," that indicates the ease with which organisms can learn to perceive contingencies. In essence,  $Q_L = 0$  for contra-prepared relations. However, the extent to which the source of this phenomenon is biological and could be overcome by relevant environmental contingencies is controversial.

If  $Q_B$ ,  $Q_2$ , and  $Q_L$  are all necessary for the perception of a causal relation, they should combine multiplicatively in affecting gross strength, since if any variable is 0, so is gross strength. On the other hand, since the other variables in Table 3 are not necessary, their effect on gross strength should be compensatory. Indeed, our review of the literature (see below), suggests the following functional form for relating gross strength to the cues,

$$\mathbf{s}(\mathbf{X},\mathbf{Y}) = \mathbf{Q}_{2}\mathbf{Q}_{\mathbf{B}}\mathbf{Q}_{\mathbf{L}}(\lambda_{\mathbf{L}}\mathbf{Q}_{\mathbf{L}} + \lambda_{1}\mathbf{Q}_{1} + \lambda_{4}\mathbf{Q}_{4}^{*}) \tag{7}$$

where the  $\lambda$ 's are attentional weights  $(\lambda_L + \lambda_1 + \lambda_4 = 1)$ . Note that while contiguity and congruity are not explicitly shown in the equation, they are included via their effects on  $Q_L$ . That is, since contiguity and congruity are a decreasing function of  $\lambda$ , and, since  $Q_L = \overline{C}^{\lambda}$  these cues do affect gross strength. We now consider further evidence supporting the form of equation (7).

(b) Several studies have examined how the cues trade-off. In developmental psychology, for example, Sedlak and Kurtz (1981) have reviewed research on three cues: temporal order, covariation, and contiguity. In summarizing the results of studies where these cues conflict, they stated:

When a temporal delay is introduced, only children 8 years of age or older offer reliable evidence of covariations. Specifically, the covariation use of younger children deteriorates when the covarying cause is temporally non-contiguous with the effect and they encounter problems in (1) rationalizing the delay, or (2) recognizing the constancy of the delay. Where these tasks are made difficult, a temporally contiguous (albeit inconsistent) cause is preferred. Thus we can tentatively rank order the three Humean principles. It appears that temporal antecedence supercedes spatial contiguity (Bullock & Gelman, 1979), and that temporal (and spatial) contiguity may sometimes outweight the covariation factor in children's judgments (Mendelson & Shultz, 1976; Siegler, 1975; Siegler & Liebert, 1974). (Sedlak & Kurtz, 1981, p. 763.)

Furthermore, in studies that have contrasted covariation with similarity, Shultz and Ravinsky (1977) found that 6-year olds were unwilling to label dissimilar factors as causes, even in the presence of systematic covariation. On the other hand, older children (10-12 years old) favored covariation over similarity. They also found that the relative weights given to similarity vs. temporal contiguity varied according to age; similarity outweighed contiguity for 6-year olds, but older children favored contiguity over similarity. In studies involving adult subjects, Einhorn and Hogarth (1982a) varied covariation, contiguity, and similarity in a factorial design. In one experiment, covariation traded-off with similarity but there was no effect for contiguity. However, when contiguity was made more salient in the experimental stimuli, it did trade-off with covariation and similarity thereby indicating the effects of attention as captured by the  $\lambda$ 's in equation (7). Finally, it is interesting to note that conflicts between similarity (congruity) and contiguity can sometimes be resolved by distinguishing between "precipitating" and "underlying" causes. The former is generally

some action or event that is high in temporal and spatial contiguity but low in similarity of length or strength with the event. The latter is generally based on high similarity of length and strength, with contiguity being less important. For example, the precipitating cause of World War I was an assassination in Sarajevo, but the underlying cause(s) were economic upheaval, German nationalism, and so on.

An important aspect of equation (7) is that it includes both the necessity of certain cues and trade-offs amongst others. This feature of our model helps to integrate the views of those who have argued both for and against the priority of cues-to-causality. On the one hand, researchers such as Shultz (1982) have presented convincing evidence that children will ignore certain cues-to-causality when these conflict with a perceived link between cause and effect  $(Q_{T_i} > 0)$ . Shultz interpreted his results as indicating that the rules governing attributions of causality give precedence to notions of "generative transmission" over the (Humean) cues-to-causality. On the other hand, we believe that posing these issues as a controversy between the Humean or "regularity" approach to causality and the "generative" theory is misplaced. First, we stress the notion that the cues-to-causality are not "rules;" they are imperfect, probabilistic indicators (Brunswik, 1952; Campbell, 1966). Thus, whereas the presence of the cues can strengthen judgments of probable cause, they do not establish it as such. Indeed, the discovery of new causal mechanisms often involves thinking in ways that counteract some of the cues. (This is discussed further below.) Second, since the cues play a crucial role in delimiting the particular models of "generative transmission" that people use in causal reasoning, it cannot be said that the cues per se are disregarded in favor of the latter. Third, while our approach puts considerable emphasis on generative transmission via

 ${\bf Q}_{\rm L}$ , it also allows for trade-offs if  ${\bf Q}_{\rm L}$  is above a threshold level. Therefore, if the Human principles are given a probabilistic interpretation and gross strength is comprised of both compensatory and noncompensatory aspects, we see considerable complementarity between the two approaches.

While "generative" links are represented in equation (7) by  $Q_{T_c}$ ,  $Q_{T_c}$  can also be taken to represent the strength of any theory (whether acquired via experience or "innate") that people bring to bear in causal reasoning. Thus,  $\mathbf{Q}_{\mathrm{L}}$  can be likened to a schema that the person brings to bear in a particular situation (Schank & Abelson, 1977; Abelson, 1981). Such schemas are usually considered to be based on past experience and world knowledge and have been studied from a variety of viewpoints. (See also the growing literature on "story grammars," Stein & Glenn, 1979; Trabasso et al., 1984; Trabasso & Sperry, 1984.) As the reader will note, we have allowed  $Q_{T_{\rm c}}$  to enter equation (7) twice; once as a necessary condition (outside the parentheses), and once in a form that allows trade-offs with both covariation and physical similarity. This form of equation was chosen to reflect the considerable literature summarized by Nisbett and Ross (1980) under the heading of "theory driven vs. data driven" judgments of covariation. In short, Nisbett and Ross show that there is much evidence to suggest that people will often allow their prior conceptions of relations (i.e.,  $Q_L$ ) to outweigh considerations of more data-driven estimates of covariation (i.e.,  $Q_1$ ). Thus, even if  $\lambda_1 Q_1$  is zero, strong causal relations can still be perceived. In analogous fashion, Shweder (1977) has summarized much literature that goes under the heading of "illusory correlation." Equation (7) allows for this phenomenon since judgments of gross strength can be high if the lack of covariation is compensated for by prior theory (i.e.,  $Q_{\rm L}$ ) and/or physical similarity between the variables (i.e.,  $Q_A^i$ ).

The tension between "theory" and "data" (i.e.,  $Q_L$  and  $Q_1$ ) that is captured in (7) is also helpful for understanding a perennial methodological issue: spurious correlation. The existence of this concept suggests that some correlations are more (or less) causally related than others, and thereby raises the issue of how to tell the difference (cf. Simon, 1954). For example, consider the correlation between the number of pigs and the amount of pig-iron (Ehrenberg, 1975). Such a correlation seems spurious when the common causal factor, "economic activity," is considered. On the other hand, consider the correlation between amount of rain and number of auto traffic accidents in a city, over the course of a year. Such a correlation does not seem spurious (or at least, less spurious). What is the difference between these two cases?

If one makes use of equation (7), the spuriousness of the correlation between pigs and pig-iron becomes apparent. That is, although the covariation cue points to a causal relation, temporal order cannot be used to specify which variable is cause or effect  $(Q_2=0)$ , and, most people cannot construct a causal chain linking pigs to pig-iron  $(Q_L=0)$ . Indeed, the judgment that the relation is spurious is made easily. Now consider the second case: assuming that there is a statistical relation  $(Q_1>0)$ , note how the other cues reinforce that link. The temporal order of rain and accidents is clear; it is relatively easy to construct a chain linking the controllability of cars on slippery roads (as long as there is high contiguity in time and space between rain and accidents); and, rain is not a standing state or condition. There seems less doubt that the correlation is "real."

When cues-to-causality conflict, spurious correlation is not the only outcome; e.g., a low or zero statistical correlation could mask a true so sal relation (Einhorn & Hogarth, 1982b). To illustrate, imagine that we were

ignorant as to the cause of birth. However, it has been suggested that sexual intercourse is related to pregnancy and the following experiment was designed to test this hypothesis: 100 couples were allocated at random to an intercourse condition, and 100 to a non-intercourse condition. As indicated in Table 4, 25 females became pregnant, and 175 did not. In light of our current

# Insert Table 4 about here

knowledge (but unknown to our hypothetical selves), we can state that the 5 people in the no-intercourse/yes-pregnancy cell represent "measurement error," i.e., faulty memory in reporting, lying, etc. Since the statistical correlation is small (r = 0.34), we might question whether the hypothesis is worth pursuing. Indeed, if the sample size were smaller, the correlation might not even be "significant." Moreover, even with a significant correlation,  $r^2 = 0.12$ , which is hardly a compelling percentage of the Y variance accounted for by X.

There are two important implications of this example. First, whereas statistics texts correctly remind us that correlation does not necessarily imply causation, the imperfect nature of this cue-to-causality is also reflected in the statement: causation does not necessarily imply correlation. We have somewhat facetiously labeled examples of the latter as "causalations," giving them equal standing with the better-known and opposite concept of spurious correlation. Second, causalation demonstrates that sole reliance on a single cue, such as covariation, is inadequate for understanding causal relations. Indeed, as equation (7) shows, gross strength can be non-zero even if  $Q_1$  is low. Therefore, the use of multiple cues highlights the role of judgment in interpretating data (see also Simon, 1954) and equation (7) provides a basis for understanding how this is done.

The examples cited so far have all involved physical causation. However,

TABLE 4

Data Matrix for Hypothetical
Intercourse-Pregnancy Experiment

		Preg / Yes	nancy No	
Intercourse	Yes	20	80	100
	No	5	95	100
	_	25	175	200

our framework can also be applied to judgments of probable cause in the social domain. Consider, for example, judgments of probable cause where actions are explained by motivations, intentions, or goals. In these cases, once the attributor postulates a motive, note that the motive has the appropriate temporal order (since the existence of the motive is assumed to precede the act), high temporal and spatial contiguity are assured, and the judgment often requires constructing only a simple link model to go from the existence of the motive to the action. Our analysis therefore suggests that in distinguishing between causal candidates, judgments of probable cause in the social domain will depend primarily on two cues, covariation and the salience of X as represented by the degree to which it is a difference-in-the-background (i.e.,  $Q_{\rm R}$ ). Indeed, it is instructive to note that both covariation and salience have been the subject of intense investigation in the literature on social attribution (Hastie, 1983; Harvey & Weary, 1984). Kelley's (1967, 1973) influential ANOVA model, for example, was constructed solely on the basis of covariation patterns.

### Role of specific alternatives

The importance of specific alternative explanations in assessing the strength of causal candidates has been stressed by many. Of particular importance is the work of Campbell and colleagues on threats to "internal validity" (Campbell & Stanley, 1963; Campbell, 1969; Cook & Campbell, 1979). Indeed, the assessment of internal validity, whereby one asks what factors other than X could have produced Y, is a basic component of causal inference. Similarly, attribution theorists (e.g., Kelley, 1973; Jones, 1979) have discussed the discounting of a causal explanation on the basis of plausible alternatives. From our perspective, specific alternatives are important beyond their obvious role in lowering the covariation between X and Y (by

increasing the frequency of the C cell). The reason is that specific alternatives allow one to use the cues-to-causality to assess their plausibility (i.e., determine the gross strength of alternatives). Hence, the content knowledge associated with a specific alternative affects its gross strength (via equation (7)), which in turn is used to discount the original explanation.

The notion of a discounting process provides an important connection between research on causal reasoning and probabilistic inference since much of the latter concerns how people update their present beliefs on the basis of new information. Therefore, the discounting of a causal explanation by specific alternatives involves a process of updating one's (causal) beliefs on the basis of new, and in this case, negative evidence (i.e., alternative explanations). Moreover, the idea that judgments of probable cause result from an updating process focuses attention on the dynamic processes underlying judgment and choice (Hogarth, 1981). Indeed, we now discuss several issues that follow from considering judgments of probable cause as resulting from an updating process.

To begin, recall equation (1), in which the net strength of a causal explanation after k specific alternatives,  $S_k(X,Y)$ , increases with the gross strength of X,  $s_0(X,Y)$ , and decreases with the gross strengths of alternatives  $Z_k$ ,  $s(Z_k,Y)$ . To illustrate the substantive issues involved in specifying the discounting process, consider the following model:

$$S_{k}(X,Y) = S_{k-1}(X,Y) - w_{k} s(Z_{k},Y)$$
 (8)

where,  $\mathbf{w}_k$  is a weight that reflects the importance given to the  $\mathbf{Z}_k$  alternative (to be discussed below). Equation (8) can be interpreted in a variety of ways; e.g., a sequential anchoring-and-adjustment model, a "Bayesian-like" update model, etc. From the present perspective, equation (8) is useful

because it raises three general issues: (a) Why is the model subtractive in form, rather than a ratio?; (b) How is the plausibility of an alternative affected by other alternatives, as well as by its similarity to X?; (c) What factors affect the importance weight, w?

(a) Consider a situation in which there are no specific alternatives to explanation X. A ratio model of the form,

$$s_{k}(x,y) = \frac{s_{k-1}(x,y) - w_{k} s(z_{k},y)}{s_{k-1}(x,y)}$$
(9)

would always yield  $S_k(X,Y) = 1$ , which is clearly unacceptable. Indeed, an important implication of the subtractive form is that the net strength of an explanation can be low even though specific alternatives do not exist (when gross strength is low). On the other hand, if gross strength is high and few specific alternatives exist, the explanation will be seen as a highly probable cause even if the inference is based on a one-shot case study (see Campbell, 1975, for an illuminating discussion of this issue). As a case in point, consider the occurrence of a huge explosion near Los Alamos, New Mexico, in July 1945. No one doubted this to be the effect of detonating an atomic bomb. Clearly, inferring causality in this poorly designed experiment was not difficult whereas assessing causality in the most meticulously designed experiments in social science is often problematic at best. When one considers why the causal inference is so strong in the bomb example, first note that there is a good link for connecting the events (atomic physics) and the cues-to-causality strongly point to a causal relation. Second, ask yourself the following question: "Would an explosion of such magnitude have occurred if an atomic bomb had not gone off?" While it is possible to think of alternative explanations for the explosion, they are so unlikely as to be

virtually non-existent. Therefore, a good explanation has high gross strength and few plausible alternatives.

- (b) A major limitation of the model represented by equation (8) concerns the similarity or redundancy of alternatives to the original explanation and to each other. For example, alternative explanations that are either more specific or more general instances of X should not discount X. Similarly, imagine that one has many alternative explanations that are highly redundant. To treat each alternative as if it were an independent explanation would clearly be incorrect. Indeed, an analogy to multiple regression with correlated variables, or conditional dependence in Bayesian statistics, is useful here. That is, the strength of a variable is seen as its incremental contribution to explaining Y when partialling out (or otherwise holding constant), other correlated factors. In the same way, the plausibility of an alternative can depend on other alternatives as well as its similarity to X. Put more formally, the plausibility of alternative  $\mathbf{Z}_{\mathbf{k}}$  needs to be expressed conditionally on X and the other alternatives; i.e.,  $S(z_k,Y|X,z_1,z_2,\ldots,z_{k-1})$ . The descriptive questions concerning how, and to what extent, people handle the redundancy between specific alternatives have barely been asked.
- (c) The importance weight in equation (8) can reflect two classes of factors: those that characterize individual differences in the way alternatives are utilized; and, process variables regarding how the discount mechanism incorporates attention, surprise, serial position effects, and the like. Consider the former and imagine that one has a very strong belief that X causes Y such that specific alternatives are weighted zero ( $w_{\rm k}=0$ , for all k). Such a person's beliefs would be impervious to negative evidence in the form of new alternatives. On the other hand, another person may be more receptive to alternatives and weight them heavily, thereby discounting the

original belief substantially. While the second person would be closer to the Popperian ideal than the first (seeking to disconfirm rather than confirm one's hypotheses), it is to be expected that people will vary considerably in the degree to which they engage in discounting.

The second class of factors can be incorporated via the importance weight in a variety of ways. For example, Einhorn and Hogarth (1983) included a contrast or surprise component in the discount process by making the importance weight a function of net strength at the k-1 level; i.e.,

$$s_k(x, y) = s_{k-1}(x, y) - w_{k-1} s(z_k, y)$$
 (10)

The model assumes that as net strength decreases after each specific alternative, the importance weight also decreases. This implies that an alternative of given strength will discount a strong explanation more than a weak one (i.e., the bigger they are, the more they fall). On the other hand, a weak causal candidate cannot be discounted much since it is already low, indeed, if X were worthless, there would be no discounting. The model was tested in several experimental situations with one and two specific alternatives and provided a good fit to the data. From the present perspective, such models open the way for incorporating various cognitive processes (such as attention and memory) in the sequential updating of causal beliefs. Indeed, the importance of viewing discounting within an updating framework lies in linking basic cognitive processes to causal inference in general and judgments of probable cause in particular.

### Multiple causation, covariation, and conjunction effects

The framework presented thus far represents what might be called the "univariate" case; that is, a single causal candidate X, and single effect Y. The difficulties of delineating the "multivariate" case are many; however,

since multiple causation is crucial to many real-world inferences, we focus on one aspect that is particularly germane to the conflict between causal and probabilistic thinking. Specifically, Tversky and Kahneman (1983) have shown that people often judge the probability of a conjunctive event as being greater than the probability of one of its constituents. From a statistical viewpoint, this is an error since a conjunction cannot be more probable than one of its components. Accordingly, Tversky and Kahneman label this response a "conjunction fallacy" giving numerous reasons for its occurrence. Two recent papers, however, raise questions about the generality of conjunction effects in causal explanations.

In the first paper (Leddo, Abelson, & Paget, in press), subjects were presented with vignettes about individuals engaged in various activities and were asked to judge the probability of both single and conjunctive reasons for why the behavior occurred. While conjunctive effects were generally found (i.e., explanations with more reasons were judged as being more probable than those with fewer reasons), they were not found when the story involved an actor not doing something (e.g., why didn't Fred stop at the Italian restaurant while he was driving down the highway?). Leddo et al. argue that in some situations, the judged probability of an explanation may increase with the number of reasons up to a point after which it declines. This notion of "single-peakedness" (Coombs & Avrunin, 1977) over the number of reasons in an explanation is an intriguing idea that we consider below. In the second paper, Locksley and Stangor (in press) presented subjects with either a rare event (suicide) or common event (getting married) and asked them to rank 10 explanations in terms of the probability that the event was caused by that explanation. The 10 explanations consisted of both single reasons and conjunctions. The results showed substantial conjunction effects when the

outcome was rare, but considerably fewer effects when the outcome was common.

To discuss conjunction effects within our framework, we assume that in the absence of other cues (or holding them constant), judgments of probable cause will be based on the covariation cue. We further assume that all cells in the covariation table receive a non-zero weight (i.e.,  $\beta_1 > 0$ ) such that  $Q_1$  is at least monotonic with statistical correlation. Now consider what happens when two factors, each positively related to Y, are conjoined into a single explanation (denoted as  $(X_1 \cap X_2)$ ). The scenarios that comprise the covariation of  $(X_1 \cap X_2)$  with Y are shown in Table 5. As was the case

# Insert Table 5 about here

with single factors, a represents the minimally sufficient scenarios that contain the various combinations of Y and  $\ddot{Y}$  with  $(X_1 \cap X_2)$  and  $(\overline{X_1 \cap X_2})$ . There are several things to note about Table 5 in comparison with Table 1: (1) The number of scenarios in which  $(X_1 \cap X_2)$  conjoins with other conditions to produce Y (cell A), must be less than or equal to the lesser of  $\alpha_{Y \cap X_a}$ and  $\alpha_{Y \cap X_2}$ . This must hold because  $\alpha_{Y \cap X_1 \cap X_2}$  is a subset of the scenarios in which  $X_1$  and  $X_2$  each combine with Y. However, by adding more valid causes to the scenario, one also decreases the number of scenarios in which  $\vec{Y}$  occurs with  $(X_1 \cap X_2)$  (cell B). Indeed, the conjunction  $(X_1 \cap X_2)$  is more fully sufficient for Y since the uncertainties associated with the conditionality of causation are reduced. Therefore, although cell A can decrease with conjunctive explanations, the B cell must show a larger decrease for conjunctive effects to occur; (2) The base rate (or marginal probability) of the event Y is a constant for any given situation. Therefore, if A and B decrease due to a conjunctive explanation, C and D must increase. This means that adding more reasons to an explanation increases its sufficiency for Y at the "cost" of decreasing its necessity. Hence, the nature of the conflict

■マンドンスのは、このではなからは、アンマンシンである。 「ア・アンジストン

TABLE 5 Covariation of Y with  $x_1 \cap x_2$ 

	Y	Ÿ	
x <sub>1</sub> nx <sub>2</sub>	α <sub>Υ</sub> ∩χ <sub>1</sub> ∩χ <sub>2</sub> (A)	α <sub>Ψ</sub> Ω <sub>1</sub> Ω <sub>2</sub> (B)	A + B
$\overline{\mathbf{x_1}^{\cap \mathbf{x}_2}}$	$\alpha_{\underline{Y} \cap (\overline{X_1 \cap X_2})}$	$\alpha_{\overline{Y}} \cap (\overline{x_1 \cap x_2})$	C + D
	A + C	B + D	A + B + C + D

that is evoked when reasons are added to an explanation results from the net effect on covariation of decreases in cell B vs. increases in cell C. Moreover, since covariation judgments depend on the relative weight given to cells B vs. C (via causal questions) as well as the differential rates of change in B and C (which depends on such factors as the base rate of Y, the base rates and intercorrelations between the various X's, etc.), a full account of conjunction effects would require a complex analysis in its own right.

However, from our perspective, the basic point is that the conflict between sufficiency and necessity can produce judgments of probable cause that are either monotonic or single-peaked with respect to the number of reasons in an explanation.

How does the above framework account for the Leddo et al. (in press) and Locksley and Stangor (in press) results? In the first study, conjunction effects were not found when an actor didn't do something (Fred didn't stop at the Italian restaurant). We would argue that the base rate of not stopping at the restaurant is very high and the various reasons (wasn't hungry, had no money, dislikes Italian food) are each sufficient (or close to sufficient) for not stopping. When one conjoins these reasons (he didn't stop because he wasn't hungry and he had no money and he doesn't like Italian food), one creates a redundant sufficient condition; i.e., a scenario that is still sufficient if a conjunct is dropped. Such a scenario is less necessary for Y since Y's occurrence implies that any single conjunct or pair of conjuncts could have happened. In order to see how this reduces overall covariation, consider the hypothetical data in Table 6.

## Insert Table 6 about here

In Table 6a, both  $X_1$  and  $X_2$  are each nearly sufficient for Y (the two correlations with Y are .14). In Table 6b, the conjunction  $(X_1 \cap X_2)$ 

TABLE 6

Hypothetical Examples of Covariation Patterns with Conjunction Effects

(a) Stop  $(\bar{Y})$ Don't stop (Y) Not hungry (x<sub>1</sub>) 1 50 49 No money (X2) r = .14Hungry  $(\bar{x}_1)$ 50 46 Money  $(\bar{X}_2)$ 95 5 100 (b) Ÿ Y  $x_1 \cap x_2$  $(\overline{x_1} \cap x_2)$ 20 0 20 r = .1175 5 80 95 5 100

is sufficient for Y, but the large increase in the C cell reduces the correlation to .11. In Table 6c,  $(X_1 \cap X_2 \cap X_3)$  makes up a redundant sufficient condition since  $(X_1 \cap X_2)$  is already sufficient. Again, the C cell increases (and is not off-set by a decrease in the B cell), and the correlation goes down (r = .08). Clearly, in this situation, increasing the number of reasons for an event leads to a monotonic decline in judged probability. Of course, we could have changed the numbers in Table 6b so that the explanation with two reasons was more likely than either the one or three-reason explanation (yielding a single-peaked function). However, the point of the example is not to argue for a particular function in all cases; it is to show how changes in the various cells, when constrained by a particular base rate, limit the nature and size of conjunction effects. Indeed, the example illustrates that conjunction effects are less likely to occur when the event of interest has a high base rate. This is consistent with our analysis since the B cell cannot be reduced much (since it is already small), relative to the potential increase in the C cell. Thus the empirical findings of few conjunction effects when base rates are high concur with the implications of our framework.

Now consider rare events that one believes to result from the conjunction of many reasons. Note that by definition, a rare event must have small A and/or C cells and conversely, large B and/or D cells. When multiple reasons are added to an explanation, the effect is likely to lower a large B cell substantially while the C cell cannot be increased very much. Therefore, increasing the reasons for a rare event will likely lead to an increase in covariation, judged probability, and conjunction effects. Again, this is consistent with empirical findings (Locksley & Stangor, in press). However, we would expect a limit to the number of reasons one can add to an explanation beyond which it is judged less probable. As far as we know, this conjecture has not yet been put to empirical test.

### DISCUSSION

Our framework for studying probable-cau: -judgments has three major components: (1) the causal background; (2) the cues-to-causality; and (3) discounting via specific alternatives. Our discussion therefore centers on the research questions and opportunities that are raised by considering these aspects of causal judgments.

The causal background. In our view, one of the most challenging problems in causal inference is to develop a theoretical model to predict the particular causal background a person is likely to adopt in a given situation. That is, whereas our framework can be used to understand the relative strength of causal candidates conditional on a given background, we are unable to make statements about how people come to focus on specific backgrounds. This predicament, however, is common to other areas of cognitive psychology. Two immediate parallels are the work by Simon and Hayes on "problem isomorphs" (1976), and that of Tversky and Kahneman on "decision frames" (1981). In common with the effects of the causal background, these investigations demonstrate how different representations of structurally identical stimuli can have important impacts on responses (for a more general review, see Hogarth, 1982). What makes these problems particularly difficult to study is that the meaning people give to stimuli is dependent on prior knowledge and experience. For example, whereas it is easy to say that people are responsive to deviations, such deviations are often defined relative to expectations; thus, any experimental work has to deal operationally with controlling these. Further confounding the issue is the fact that once a person has adopted a particular field, the cues-to-causality are likely to reinforce the initial viewpoint and thus the person's understanding of the situation.

Although the determination of the causal background remains problematic,

consider the distinction between causes and conditions made earlier. That is, in discussing how causal variables in one background can become conditions in another, we stated that causal factors are generally intrusive or abnormal events rather than standing states or normal events. However, standing states can be seen as causal if the outcome to be explained is a standing state. For example, what is the cause of "poverty?" Since the effect to be explained is a standing state of large duration and magnitude, the congruity cue demands that the cause be of comparable duration and magnitude. Indeed, to suggest a simple cause is to be accused of being "simplistic," only "root" or "underlying" causes will do. Therefore, standing states can be causal if the need to maintain congruity is important. However, even in this case, note the subtle effect of the causal background in the posing of the question; i.e., poverty requires an explanation because it is seen as a deviation from some desired state. On the other hand, if poverty were seen as a usual state, one would be asking, what causes prosperity? Therefore, even when standing states are viewed as causal, we would argue that there is an implicit deviation in the posing of the question.

Finally, the role of deviations in a background goes beyond its importance in causal inference. For example, when expectations that rest on an assumed background are violated, surprise can be an important cue for reorganizing or restructuring one's hypotheses and explanations. Imagine a hit-and-run accident in which all the witnesses said that the offending car was going 73 miles per hour at the moment of impact. Since we expect much greater variability in such estimates, as well as round numbers, this surprising unanimity might cue one to ask whether the witnesses had colluded in their responses. Similarly, the structure of outcomes can suggest new hypotheses such that the diagnosis contradicts the surface meaning of the

evidence. Thus, scientific data that are too perfect can suggest fraud (see, for example, Kamin, 1974, on Burt's twin data; Bishop, et al, 1975, on Mendel's pea experiment), evidence in a trial that is too consistent and obvious can suggest the defendant was "framed," and one can "protesteth too much" in a variety of circumstances. Such examples illustrate that violations of expectations can trigger a re-structuring of alternatives. Of course, specifying the conditions that lead to re-structuring as opposed to other responses remains an important and unanswered question.

The cues-to-causality. In discussing the cues-to-causality and how these combine with the background, we organize our comments around three topics:

(a) the functions the cues perform for the organism and the attendant costs and benefits; (b) unresolved, and possibly unresolvable issues concerning the cues; and (c) opportunities for further research.

(a) The object of establishing causal relations is to make sense of the world so that the organism can achieve its goals. From this perspective, both the adoption of particular causal backgrounds and the use of the cues-to-causality are remarkably efficient mechanisms in that they reduce the scope of the inferential task within limited information processing constraints. That is, the field delimits the focus of attention, and the cues constrain the types of causal relations constructed (cf. Table 2). Moreover, given the inherent complexity of the environment, one can also argue that there are functional advantages to both the fluctuating nature of attention, and the probabilistic character of the cues. Specifically, fluctuations in attention permit alternative representations of the same or similar tasks (see above), and probabilistic cues imply that one need not always construct the same scenarios within a field. However, we note three important trade-offs that are implicit in these inferential mechanisms: (i) "order-out-of-chaos" vs.

creativity. As just noted, the causal field and the cues-to-causality play important roles in limiting the number of interpretations people make in inferential tasks, and thus in creating "order-out-of-chaos." Furthermore, the adoption of a particular background and the use of the cues proceed quickly and are often marked by a lack of awareness that a delimiting process has taken place. The benefits to be gained from such automatized processes are large. However, they come at the cost of reducing the probability that people can achieve more creative representations of inferential tasks. Indeed, Campbell (1960) has stressed the importance of deliberately introducing random variation to stimulate creative efforts, especially in science. Without such random perturbations, he argues that the forces that maintain a person's particular conception of a problem are too strong. Moreover, the literature on creativity has many examples of techniques that are aimed precisely at making people aware of the delimiting assumptions they bring to tasks (e.g., Adams, 1976). In addition, when using such techniques, people are often requested to refrain from counterfactual reasoning and to make specific use of analogies and paradox to enjoin previously disconnected ideas. In short, to restructure problems in creative ways frequently requires attempts to counter the habitual forces of causal reasoning; (ii) acquiring superstitions vs. causal knowledge. Since the cues are probabilistic indicators of causal relations, it follows that they will sometimes indicate invalid causal relations. Moreover, these relations could well be reinforced by environments in which they cannot be adequately tested (cf. Einhorn, 1980). Thus, the cues-to-causality can also lead to the acquisition of superstitious beliefs (cf. Skinner 1966); (iii) imagination vs. uncertainty. By constraining the interpretation of information, the cues also reduce feelings of uncertainty in inference. However, given the probabilistic nature of the

cues, the level of uncertainty experienced could increase if people exercised imagination in constructing alternative causal scenarios that were either consistent with or even violated the cues. In this sense, therefore, the cues mediate the trade-off people make between exercising imagination and experiencing feelings of uncertainty.

(b) We have assumed throughout the paper that the cues-to-causality have imperfect but non-zero ecological validities, i.e., each cue is predictive of a true causal relation. How do we know this? Simply put, we don't. The reason is that without some measure of "true" causality, no determination of accurate causal knowledge is possible. However, the fact that the cues we have considered are implicated in a wide variety of studies with both human and animal subjects, leads us to believe that they would not continue to be used if they were useless. Therefore, our argument is a functional and practical one; viz., given the importance of learning and inferring causal relations for survival, we do not believe that the cues on which this depends are totally worthless. On the other hand, we do not advocate the position that if something is used, it must be beneficial to the organism. Such a position is untenable for many reasons (see Einhorn & Hogarth, 1981).

Second, we have also argued that the cues are partially redundant (intercorrelated) and that this affects the inference process. However, to show how, and to what extent the cues are correlated would require an elaborate (and problematic) ecological analysis that is beyond the scope of this paper. Nevertheless, the assumption of correlated cues seems warranted since people have strong expectations concerning what cues go together. Indeed, just as in the perception of incomplete figures (where one fills in the missing parts), scenarios are filled in by assuming that cues not explicitly mentioned are present. Thus, the fact that one generally perceives the world as coherent,

suggests that the cues-to-causality are redundant to some degree.

A third problematic issue raised by our framework concerns the origins of causal reasoning. What are the relations between the cues, schemas, and the notion that people seem to need to perceive the transfer of causal "energy" via a link mechanism prior to making attributions of cause? As argued by Cook and Campbell (1979), amongst others, the need to perceive cause has important evolutionary significance in environments where the discovery of causal relations has high survival value. Furthermore, as noted earlier, we believe that Seligman's (1970) analysis showing how both humans and animals are "contra-prepared" to learn certain types of contingencies does suggest some genetic predispositions toward certain kinds of causal attributions. If one concedes this point, and accepts the fact that some part of causal reasoning is as yet "unknowable," we believe that the role of the cues and their relation with schemas is fairly straightforward. That is, the cues both constrain the causal schemas people can imagine and, in an important sense, are the building blocks of such schemas. One interesting aspect of schema construction lies in how schemas change as people acquire greater knowledge over time. At one extreme is the tendency to anthropomorphize phenomena one does not understand. For example, consider how small children tend to ascribe personalities to the elements (e.g., the wind) so that they can explain changes in the environment by attributing intentions to physical forces (see e.g., Piaget, 1974). Moreover, note, as discussed earlier in reference to social causation, that as soon as one attributes intentions, motives, and goals, causal reasoning is considerably facilitated since the constraints implied by the cues-to-causality are automatically respected. However, over time such anthropomorphic explanations are dropped in favor of accounts framed in terms of physical causation. Thus, mature adults are often skeptical of

explanations of "psychic" phenomena unless they are couched in terms of models involving the transmission of some form of energy. Indeed, it is perhaps paradoxical that adult believers in ESP tend to be found either among the highly credulous or scientifically highly sophisticated segments of the population. However, the bases of the beliefs in the two groups are quite different. That is, whereas the former are prepared to believe in invisible mystical forces, the latter also postulate invisible forces but deny that they are mystical.

(c) Our framework suggests several avenues of research concerning the cues and how these combine with the background in affecting gross strength. First, whereas we believe that equation (7) is a useful summary of the existing literature, the functional form of the equation both needs to be tested and suggests several explicit hypotheses. For example, would the necessity of the temporal order cue be maintained if there were some doubt as to the relative timing of potential cause and effect? Can one demonstrate experimentally the effects of salience via the background cue in the hypothesized manner? The hypothesized form of the link variable  $(Q_T)$  suggests experiments in which one systematically varies both contiguity (thus affecting the number of links) and covariation at the link level. Second, our framework places great emphasis on how the background and cues constrain the construction of causal scenarios. However, it should also be possible, within the framework, to explore systematically factors that affect the particular scenarios people select from the "feasible" sets of scenarios. Different levels of substantive expertise, for instance, should lead to scenarios that vary in complexity as measured by the number of links in the causal chain. Experts, one will recall, view causal mechanisms in more reductionistic terms, i.e., their field differs from novices. Alternatively, one could also examine

the effects of attention by making some information more salient than others. Einhorn and Hogarth (1982a), for example, found that judgments of causal strength could be affected by making the cue of contiguity differentially salient in experimental stimuli. However, they did not explore the nature of the causal schemas people used in the two conditions. Moreover, to understand these kinds of issues researchers will have to go beyond eliciting data via judgments on rating scales but use more data-intensive collection techniques such as verbal protocols. As revealed in our analysis of the covariation cue, the manner in which a causal question is asked (e.g., emphasizing the conditionality or multiplicity of causation) could well affect judgments of causal strength by emphasizing particular cells of  $2 \times 2$  covariation matrices at the expense of others. Moreover, one could argue that this phenomenon is more likely to occur in situations involving social as opposed to physical causation. The reason is that attributions of social causation that use concepts such as goals, motives, and intentions are particularly sensitive to the covariation cue and operate with short links between cause and effect. In many situations involving physical causation, however, if the intermediate scenarios involve several links, the relative effect of the covariation cue is reduced.

One intriguing possibility centers on incorporating the cues-to-causality in "expert systems" used in artificial intelligence (Duda & Shortliffe, 1983). Two important problems faced by designers of such systems are a lack of theoretical structures for handling diagnostic tasks and the corresponding level of detailed substantive knowledge that has to be programmed in order to compensate for the lack of such diagnostic theory. If one considers the cues as providing a set of meta-principles for guiding problem search, we see considerable advantage in using them in expert systems to determine regions

of feasible solutions. Moreover, even though we cannot demonstrate that the cues have ecological validity (see above), we can point to a successful precedent of this type. Specifically, recall how successful "bootstrapping" models have been in the area of clinical prediction even in the absence of measurable criteria (see e.g., Dawes, 1971; Camerer, 1981). At the very least, our suggestions could be used to reduce inconsistency in causal judgment (cf. Hammond, Rursch & Todd, 1964; Goldberg, 1970).

Discounting via specific alternatives. In addition to lowering the covariation between X and Y, specific alternatives point to the possibility of replacing the current explanation rather than simply disconfirming it. That is, since the goal of causal inference is to find some explanation for the observed effects, the discounting of an explanation by specific alternatives still leaves one with the question, "If X didn't cause Y, what did?" Therefore, while the testing of hypotheses via comparison with alternatives is part of the causal inference process, one is still left with finding a plausible explanation. In fact, the distinction between testing hypotheses and searching for better ones can be likened to the difference between a "disconfirmation" vs. "replacement" mode of inference. The replacement view is consistent with the Kuhnian notion that theories in science are not discarded, despite evidence to the contrary, if they are not replaced by better alternatives (Kuhn, 1962). We believe that the replacement view is equally strong in everyday inference. A useful analogy might be the following: how many people would read detective stories if the author only revealed who didn't do it? Moreover, note that replacement is more powerful than disconfirmation since the former subsumes the latter.

Given that specific alternatives play a central role in the causal inference process, how do they discount an explanation and even replace it?

Our earlier discussion focused on the former question since we are unaware of research on the latter. However, although we treated discounting as part of a more general process concerned with the updating of beliefs, several important questions remain. For example, earlier we discussed the issue of the redundancy of explanations and the difficulty of incorporating this factor into the discounting process. A further complication concerns how the redundancy of explanations is affected by their generality. For example, in decisionmaking-under-uncertainty, the expected utility model, [E(U)], is a major theory of choice (Schoemaker, 1982). However, a number of competing theoretical positions have been developed in recent years (Kahneman & Tversky, 1979; Karmarkar, 1979; Chew & MacCrimmon, 1979; Bell, 1982). Since these theories provide alternative explanations for choices amongst gambles, evidence in their favor should discount the strength of E(U). On the other hand, the competitors can be seen as generalizations of the E(U) model and thus, evidence in their favor can be viewed as providing some support for E(U). Moreover, since the competitors have numerous structural similarities, evidence supporting them may be due to their common rather than distinguishing features. Second, while we discussed the discounting process in terms of a sequential updating model, there are many procedural factors that could affect the amount and type of discounting (see Lopes, 1982). For example, would the amount of discounting be different if multiple alternatives were considered as a unit rather than evaluated piece-by-piece?; Does the length and complexity of information affect attention such that order effects occur? If so, under what conditions is primacy more likely than recency, and vice versa? Third, the updating of causal beliefs depends on positive as well as negative evidence. Therefore, a full updating model will have to deal with both the discounting and accretion of causal strength.

#### CONCLUSION

Bertrand Russell (1948) stressed the uncertain nature of inferences in science and common sense by noting that they "differ from those of deductive logic and mathematics in a very important sense, namely, that when the premises are true and the reasoning correct, the conclusion is only probable" (1948, p. 335, emphasis in original). Since causal inference is an essential and ubiquitous cognitive activity, we have focused on research concerning how people make judgments of probable cause. In providing both a conceptual framework and review of the relevant literature, we have tried to provide links between the extensive literature on causation and the burgeoning psychological literature on judgment-under-uncertainty. In attempting this integration, we have argued that judgments of probable cause are affected by three main factors: a causal background or field, probabilistic cues-tocausality, and a discounting process for specific alternative explanations. Moreover, these general ideas can be summarized by a perceptual analogy in which causal candidates are differences-in-a-background (figures are seen against ground), good explanations arise from internally consistent patterns of cues (good figures form a "gestalt"), and good explanations have few plausible alternatives (as do good figures).

Whereas our framework accounts for much literature and leads to many testable implications, it by no means explicates all aspects of causal reasoning. In particular, inferences made on the basis of complex scenarios, defining and measuring the "coherence" of a causal explanation (Trabasso et al., 1984), issues of multiple and redundant causation, etc., present formidable difficulties and challenges for psychological research. However, given the complexity of these issues, it seems appropriate to have started with a model based on alternatives, background, and cues; i.e., the ABC of causal inference.

#### REFERENCES

- Abelson, R. P. (1981). Psychological status of the script concept. American

  Psychologist, 36, 715-729.
- Adams, J. L. (1976). Conceptual blockbusting: A pleasurable guide to better problem solving. San Francisco: San Francisco Book Co.
- Alloy, L. B., & Abramson, L. Y. (1979). Judgment of contingency in depressed and nondepressed students: Sadder but wiser? <u>Journal of Experimental</u>

  Psychology: General, 108, 441-485.
- Alloy, L. B., & Tabachnik, N. (1984). Assessment of covariation by humans and animals: The joint influence of prior expectations and current situational information. <a href="Psychological Review">Psychological Review</a>, 91, 112-149.
- Arkes, H. R., & Harkness, A. R. (1983). Estimates of contingency between two dichotomous variables. <u>Journal of Experimental Psychology</u>: <u>General</u>, <u>112</u>, 117-135.
- Bar-Hillel, M. (1980). The base-rate fallacy in probability judgments. Acta Psychologica, 44, 211-233.
- Bell, D. E. (1982). Regret in decision making under uncertainty. Operations
  Research, 30, 961-981.
- Bishop, Y. M. M., Fienberg, S. E., & Holland, P. W. (1975). Discrete multivariate analysis: Theory and practice. Cambridge, MA: The MIT Press.
- Brunswik, E. (1952). The conceptual framework of psychology. Chicago:
  University of Chicago Press.
- Bunge, M. (1979). Causality and modern science. New York: Dover.
- Camerer, C. F. (1981). General conditions for the success of bootstrapping models. Organizational Behavior and Human Performance, 27, 411-422.

- Campbell, D. T. (1960). Blind variation and selective retention in creative thought as in other knowledge processes. <u>Psychological Review</u>, 67, 380-400.
- Campbell, D. T. (1966). Pattern matching as an essential in distal knowing.

  In K. R. Hammond (Ed.), The psychology of Egon Brunswik. New York: Holt,

  Rinehart and Winston.
- Campbell, D. T. (1969). Reforms as experiments. American Psychologist, 24, 409-429.
- Campbell, D. T. (1975). Degrees of freedom and the case study. Comparative Political Studies, 8, 178-193.
- Campbell, D. T., & Stanley, J. C. (1963). Experimental and quasiexperimental designs for research. Chicago: Rand-McNally.
- Chew, S.-H., & MacCrimmon, K. R. (1979). Alpha-nu choice theory: A generalization of expected utility theory. Working paper 669, University of British Columbia, Vancouver, Canada.
- Cohen, L. J. (1977). The probable and the provable. Oxford: Clarendon Press.
- Cook, T. D., & Campbell, D. T. (1979). Quasi-experimentation: Design and analysis for field settings. Chicago: Rand-McNally.
- Coombs, C. H., & Avrunin, G. S. (1977). Single-peaked functions and the theory of preference. Psychological Review, 84, 216-230.
- Crocker, J. (1981). Judgment of covariation by social perceivers.

  Psychological Bulletin, 90, 272-292.
- Crocker, J. (1982). Biased questions in judgment of covariation studies.

  Personality and Social Psychology Bulletin, 8, 214-220.
- Dawes, R. M. (1971). A case study of graduate admissions: Applications of three principles of human decision making. <u>American Psychologist</u>, <u>26</u>, 180-188.

- Dawes, R. M. (undated). How to use your head and statistics at the same time or at least in rapid alternation. University of Oregon working paper.
- Duda, R. O., & Shortliffe, E. H. (1983). Expert systems research. Science, 220, 261-268.
- Eddy, D. M. (1982). Probabilistic reasoning in clinical medicine: Problems and opportunities. In D. Kahneman et al. (Eds.), <u>Judgment under uncertainty</u>: Heuristics and biases. New York: Cambridge University Press.
- Ehrenberg, A. S. C. (1975). <u>Data reduction: Analyzing and interpreting</u> statistical data. New York: John Wiley.
- Einhorn, H. J. (1980). Learning from experience and suboptimal rules in decision making. In T. S. Wallsten (Ed.), Cognitive processes in choice and decision behavior. Hillsdale, NJ: Erlbaum.
- Einhorn, H. J., & Hogarth, R. M. (1978). Confidence in judgment:

  Persistence of the illusion of validity. Psychological Review, 85,

  395-416.
- Einhorn, H. J., & Hogarth, R. M. (1981). Behavioral decision theory:

  Processes of judgment and choice. Annual Review of Psychology, 32, 53-88.
- Einhorn, H. J., & Hogarth, R. M. (1982a). A theory of diagnostic inference:

  II. Judging causality. Center for Decision Research, Graduate School of

  Business, University of Chicago.
- Einhorn, H. J., & Hogarth, R. M. (1982b). Prediction, diagnosis, and causal thinking in forecasting. Journal of Forecasting, 1, 23-36.
- Einhorn, H. J., & Hogarth, R. M. (1983). A theory of diagnostic inference:

  Judging causality. Center for Decision Research, Graduate School of

  Business, University of Chicago.

- Fincham, F. D., & Jaspars, J. M. (1980). Attribution of responsibility:

  From man the scientist to man as lawyer. Advances in Experimental Social

  Psychology, 13, 82-138.
- Garcia, J. (1981). Tilting at the paper mills of academe. American

  Psychologist, 36, 149-158.
- Garcia, J., McGowan, B., Ervin, F. R., & Koelling, R. (1968). Cues: Their relative effectiveness as reinforcers. Science, 160, 794-795.
- Garcia, J., McGowan, B., & Green, K. (1972). Sensory quality and integration: Constraints on conditioning. In A. H. Black & W. F. Prokasy

  (Eds.), Classical conditioning II: Current research and theory. New

  York: Appleton-Century-Crofts.
- Garner, W. R. (1966). To perceive is to know. American Psychologist, 21,
- Garner, W. R. (1970). Good patterns have few alternatives. American Scientist, 58, 34-42.
- Goldberg, L. R. (1970). Man versus model of man: A rationale, plus some evidence for a method of improving on clinical inferences. <u>Psychological Bulletin</u>, 73, 422-432.
- Goldberg, L. R. (1981). Unconfounding situational attributions from uncertain, neutral, and ambiguous ones: A psychometric analysis of descriptions of oneself and various types of others. <u>Journal of Personality and Social Psychology</u>, 41, 517-552.
- Hammond, K. R. (1955). Probabilistic functionalism and the clinical method. Psychological Review, 62, 255-262.
- Hammond, K. R., Hursch, C. J., & Todd, F. J. (1964). Analyzing the components of clinical inference. Psychological Review, 71, 438-456.

- Hart, H. L., & Honoré, A. M. (1959). Causation in the law. Oxford:
  Clarendon Press.
- Harvey, J. H., & Weary, G. (1984). Current issues in attribution theory and research. Annual Review of Psychology, 35, 427-459.
- Hastie, R. (1983). Social inference. Annual Review of Psychology, 35, 511-542.
- Hastie, R. (1984). Causes and effects of causal attribution. <u>Journal of</u>

  Personality and Social Psychology, 46, 44-56.
- Heider, F. (1958). The psychology of interpersonal relations. New York:

  John Wiley.
- Helson, H. (1964). Adaptation-level theory. New York: Harper.
- Hogarth, R. M. (1981). Beyond discrete biases: Functional and dysfunctional aspects of judgmental heuristics. Psychological Bulletin, 90, 197-217.
- Hogarth, R. M. (Ed.). (1982). Question framing and response consistency.

  San Prancisco, CA: Jossey-Bass.
- Jenkins, H. M., & Ward, W. C. (1965). Judgment of contingency between responses and outcomes. <u>Psychological Monographs</u>: <u>General and Applied</u>, 79 (Whole No. 594), 1-17.
- Jones, E. E. (1979). The rocky road from acts to dispositions. American Psychologist, 34, 107-117.
- Jones, E. E., & Nisbett, R. E. (1972). The actor and the observer:

  Divergent perceptions of the causes of behavior. In E. E. Jones et al.

  (Eds.), Attribution: Perceiving the causes of behavior. Morristown,

  NJ: General Learning Press.
- Kahneman, D., & Tversky, A. (1979). Prospect theory: An analysis of decision under risk. <u>Econometrica</u>, <u>47</u>, 263-291.
- Kamin, L. J. (1974). The science and politics of IQ. Potomac, MD: Erlbaum.

- Karmarkar, U. S. (1978). Subjectively weighted utility: A descriptive extension of the expected utility model. Organizational Behavior and Human Performance, 21, 61-72.
- Kelley, H. H. (1967). Attribution theory in social psychology. In D. Levine (Ed.), Nebraska Symposium on Motivation, 1967. Lincoln: University of Nebraska Press.
- Kelley, H. H. (1973). The processes of causal attribution. American Psychologist, 28, 107-128.
- Kelley, H. H., & Michela, J. L. (1980). Attribution theory and research.

  Annual Review of Psychology, 31, 457-501.
- Kuhn, T. S. (1962). The structure of scientific revolutions. Chicago:
  University of Chicago Press.
- Leddo, J., Abelson, R. P., & Gross, P. (in press). Conjunctive explanations:

  When two reasons are better than one. <u>Journal of Personality and Social</u>

  Psychology.
- Lipe, M. G. (1982). A cross-study analysis of covariation judgments. Center for Decision Research, Graduate School of Business, University of Chicago.
- Locksley, A., & Stangor, C. (in press). Why vs. how often: Causal reasoning and the incidence of judgmental biases. <u>Journal of Experimental Social Psychology</u>.
- Lopes, L. L. (1982). Toward a procedural theory of judgment. Wisconsin

  Human Information Processing Program, University of Wisconsin, Report 17.
- Mackie, J. L. (1965). Causes and conditions. American Philosophical
  Quarterly, 2, 245-264.
- Mackie, J. L. (1974). The cement of the universe: A study of causation.

  Oxford: Clarendon Press.
- Michotte, A. (1946). La perception de la causalité. Paris: Vrin.

- Nisbett, R. E., & Ross, L. D. (1980). Human inference: Strategies and shortcomings of social judgment. Englewood Cliffs: Prentice-Hall.
- Piaget, J. (1974). Understanding causality. New York: Norton.
- Russell, B. (1948). <u>Human knowledge: Its scope and limit</u>. New York: Simon & Schuster.
- Schank, R. C., & Abelson, R. P. (1977). Scripts, plans, goals and understanding: An inquiry into human knowledge structures. Hillsdale, NJ: Erlbaum.
- Schoemaker, P. J. H. (1982). The expected utility model: Its variants, purposes, evidence and limitations. <u>Journal of Economic Literature</u>, 20, 529-563.
- Schustack, M. W., & Sternberg, R. J. (1981). Evaluation of evidence in causal inference. <u>Journal of Experimental Psychology: General, 110, 101-120.</u>
- Sedlak, A. J., & Kurtz, S. T. (1981). A review of children's use of causal inference principles. Child Development, 52, 759-784.
- Seligman, M. E. P. (1970). On the generality of the laws of learning.

  Psychological Review, 77, 406-418.
- Shapiro, A. K. (1960). A contribution to a history of the placebo effect.

  Behavioral Science, 5, 109-135.
- Shultz, T. R. (1982). Rules of causal attribution. Monographs of the Society for Research in Child Development, 47, 1-51.
- Shultz, T. R., & Ravinsky, F. B. (1977). Similarity as a principle of causal inference. Child Development, 48, 1552-1558.
- Shweder, R. A. (1977). Likeness and likelihood in everyday thought: Magical thinking in judgments about personality. <u>Current Anthropology</u>, 18, 637-658.

- Siegler, R. S. (1976). The effects of simple necessity and sufficiency relationships on children's causal inferences. Child Development, 47, 1058-1063.
- Siegler, R. S., & Liebert, R. M. (1974). Effects of contiguity, regularity, and age on children's causal inferences. <u>Developmental Psychology</u>, 10, 574-579.
- Simon, H. A. (1954). Spurious correlation: A causal interpretation.

  Journal of the American Statistical Association, 49, 467-479.
- Simon, H. A., & Hayes, J. R. (1976). The understanding process: Problem isomorphs. Cognitive Psychology, 8, 165-190.
- Skinner, B. F. (1966). The phylogeny and ontogeny of behavior. Science, 153, 1205-1213.
- Smedslund, J. (1963). The concept of correlation in adults. Scandinavian

  Journal of Psychology, 4, 165-173.
- Stein, N. L., & Glenn, C. G. (1979). An analysis of story comprehension in elementary school children. In R. O. Freedle (Ed.), <u>Advances in discourse processing</u>: New directions in discourse processing, 2. Norwood, NJ:

  Ablex.
- Strawson, P. F. (1952). Introduction to logical theory. London: Methuen.
- Suppes, P. (1970). A probabilistic theory of causality. Amsterdam: North-Holland.
- Susser, M. (1973). Causal thinking in the health sciences. New York:
  Oxford University Press.
- Toda, M. (1977). Causality, conditional probability and control. In

  A. Aykac & C. Brumat (Eds.), New developments in the application of

  Bayesian methods. Amsterdam: North-Holland.

- Trabasso, T., Secco, T., & Van Den Broek, P. (1984). Causal cohesion and story coherence. In H. Mandl et al. (Eds.), <u>Learning and comprehension of text</u>. Hillsdale, NJ: Erlbaum.
- Trabasso, T., & Sperry, L. L. (1984). The causal basis for deciding importance of story events. Department of Education, University of Chicago.
- Tversky, A. (1977). Features of similarity. <u>Psychological Review</u>, 84, 327-352.
- Tversky, A., & Kahneman, D. (1980). Causal schemas in judgments under uncertainty. In M. Fishbein (Ed.), Progress in social psychology,

  Vol. 1. Hillsdale, NJ: Erlbaum.
- Tversky, A., & Kahneman, D. (1981). The framing of decisions and the psychology of choice. Science, 211, 453-458.
- Twersky, A., & Kahneman, D. (1983). Extensional versus intuitive reasoning:

  The conjunction fallacy in probability judgment. Psychological Review,

  90, 293-315.
- Waller, W. S., & Felix, W. L. (1982). The auditor and learning from experience: Some empirical evidence. Department of Accounting, University of Arizona.
- Ward, W. C., & Jenkins, H. M. (1965). The display of information and the judgment of contingency. Canadian Journal of Psychology, 19, 231-241.
- Watson, D. (1982). The actor and the observer: How are their perceptions of causality divergent? Psychological Bulletin, 92, 682-700.

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The assumption that  $\lambda_L + \lambda_1 + \lambda_4 = 1$  implies that gross strength is made up of two major parts: a multiplicative component  $(Q_B \cdot Q_2 \cdot Q_L)$  and a weighted average  $(\lambda_L Q_L + \lambda_1 Q_1 + \lambda_4 Q_4')$ . Furthermore, if  $0 < Q_4' < 1$ , the gross strength of X for Y will be appropriately bounded between 0 and 1; i.e., 0 < s(x,Y) < 1.

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